THE SURGICAL CLINICS OF JOHN B. MURPHY, M.D.

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THE SURGICAL CLINICS OF JOHN B. MURPHY, M.D.

AT

MERCY HOSPITAL, CHICAGO

AUGUST, 1912

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PHILADELPHIA AND LONDON

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ACUTE APPENDICITIS AND PNEUMONIA

HISTORY

THIS child is seven years old. He entered Mercy Hospital March 9, 1912, because of abdominal pain and tenderness. Family history is negative as to appendicitis and tuberculosis. Aside from the present trouble, he has been a very strong, rugged boy.

Present Trouble.—The patient's father states that his boy's trouble dates from the time when he was one year old. Then, and on about four or five other occasions before he was five years old, he had attacks of sickness about which the father remembers little, except that he had pain in the abdomen and vomited. He is fairly certain that his boy had fever during some of these attacks, but does not know about tenderness or chills. He was sick three or four days at each of these attacks, and between them he had no pain. For three or four years he complained of epigastric oppression and the eructation of gas. During that time he has been constipated, always worse during the attacks.

In March, 1911, patient had pain in the lower right quadrant of the abdomen. It was severe, colicky, and intermittent in nature. It was present all over the central portion of abdomen, but most severe in the region of McBurney's point. A doctor was called, and finding temperature and tenderness at McBurney's point in addition to the above-named symptoms, diagnosed appendicitis and advised operative treatment. After five days in bed the child apparently was well again.

September 28, 1911, he was again seized with pain in the right iliac region. It was more severe than it had ever been. He vomited and had tenderness over the abdomen, but was all right in three days.

During the first days of January, 1912, he had another attack of pain and emesis keeping him sick for one day.

On the morning of March 6th, three days ago, his present attack

began with severe pain generalized over the abdomen, but in a very few minutes becoming more pronounced at McBurney's point. Two hours later he vomited. Pain and emesis recurred intermittently all that day and until 3 o'clock of the morning following, when they subsided somewhat after the administration of an anodyne by a doctor. Tenderness in the right iliac region has been acute since the onset of this attack and still continues so.

On entering the hospital, March 7, 1912, temperature was 101° F.; pulse, 136. Two hours later the pulse was 160 and the temperature 102° F.

Leukocyte count, 16,800. Urine negative.

Physical Findings.—Examination of heart negative. Examination of chest revealed increased breath-sounds, bronchial breathing, and loud râles all over front of chest, but much worse on the right side.

Operation March 15, 1912.

COMMENTS AND OPERATION

Dr. Murphy: Please follow the history of this case carefully, as it varies from the regular course.

(Intern reads history.)

Dr. Murphy: What do you mean by "during the month of March"?

The Intern: Doctor, he knew it was March, 1911, but he could not tell me when it was.

Dr. Murphy: Give a distinct time in the month. "During" implies a space. If you say "in the month of March," it would mean at some time in the month of March, but "during the month of March" means he had it all through the month.

(The intern reads more of the history.)

Dr. Murphy: This boy came into the hospital on the ninth of this month, and we saw him on the evening of the ninth, when he had a temperature of 102° F.; he had bronchial râles, short and painful breathing, and we diagnosed pneumonia with acute appendicitis. He was very ill with his pneumonia. His breathing was rapid and short. His abdomen was distended. He was very sensitive in his right iliac fossa, but the operation for appendi-

citis in an acute stage of pneumonia results very unfavorably in the large percentage of the cases and is not justifiable.

We felt we had better let him get over his pneumonia and get a subsidence of his appendicitis, until he has reached the stage where we could operate with comparative safety. His pneumonia cleared up, and his temperature went down to about normal, but is now going up again. He has an area of sensitiveness and resistance which I interpret as a mass in his right iliac fossa. The temperature yesterday was 103° F.; that means he has a secondary infection of a new zone around his appendiceal abscess. It has been present during the course of his pneumonia, and he is in danger of having an acute thrombophlebitis of the portal veins and a rupture of the pus into the peritoneal cavity.

On this occasion we will make no effort at all to take out his appendix unless it has sloughed and is loose. We will endeavor to conduct the pus from that abscess to the nearest point of outlet, with the least possible soiling of his peritoneum, and nothing more. When he is finally over his attack, we will take out his appendix, for in all probability he is going to have other attacks. I remember a case of acute appendicitis we had ten days or two weeks ago, in which the appendix had sloughed entirely free and came out with the pus in two gangrenous masses.

Relative to pneumonia and pain in the right side of the abdomen, even down to the right iliac region, that is an important condition, as we have so many patients coming in to be operated on for appendicitis associated with diaphragmatic or pleuritic pains. They have pain in and fixation of the abdomen, and pronounced elevation of temperature. I recall an early experience of that kind when I first began to take out appendices. I was called 100 miles from Chicago by telephone to come immediately to see a case of intestinal obstruction. The doctor said over the phone that the patient had been sick about three days; that the abdomen was enormously distended; that he had some vomiting in the early stage; there were no bowel movements from the beginning, and it was thought he had intestinal obstruction. The doctor informed me that he had a temperature of 103° F. the first day. It can be laid down as a law that in the first days of me-

chanical obstruction of the intestine these patients never have an elevation of temperature, but late in the mechanical obstruction patients may have elevation of temperature associated with gangrene and infection. A hypodermic injection of morphin stopped the pain from which the patient was suffering. I concluded that the patient had an acute appendicitis and made the trip. On examination I found the abdomen enormously distended; the breathing much accelerated and labored, the pulse 160, and the lips cyanotic. There was a complete consolidation of the lower lobe of the right lung. There was no particular sensitiveness over the McBurney point nor in any portion of the abdomen. Friction sounds were heard all over the lower right lobe. This was a straight case of pneumonia with diaphragmatic and costal pleurisy, and the absence of bowel movements was due to the reflex paralysis of the bowel so commonly associated with this disease, and so frequently diagnosed as an abdominal lesion, such as appendicitis, cholecystitis, intestinal obstruction, peritonitis, etc. There was a reflex spasm of the intestine, and the patient had a bowel movement! Professor James Adams Allen, one of my best teachers, used to say: "What will you do when you have obstruction of the bowel and can't get a bowel movement? Give a cathartic?—yes and no. Give strychnin to stimulate peristalsis; give morphin?—yes and no. Why give morphin when you cannot get a bowel movement? Does not that stop peristalsis? Yes, but when peristalsis has ceased from reflex causes, as the passage of a ureteral calculus, hepatic calculus, diaphragmatic pleurisy, strangulation of the ovary, etc., morphin stops the reflex pain, and permits peristalsis to come on and produces a bowel movement." We will now open the abdomen over the mass in the right iliac region in the usual manner. The abscess is high up and behind the great mesentery. It formed in a very unusual position, behind the coils of the intestine, and down under the mesentery. This accounts for the great danger of pylephlebitis. Here is a place where you have to handle conditions with the greatest of care. The abscess burrowed posteriorly and behind the coils of the intestine. It is situated on the vena cava and aorta. This type of mesenteric

appendiceal abscess is described in my article on Appendicitis in Keen's Surgery.

Let the records show that there were agglutinations of the bowel; the abdomen was free from adhesions in front; the base of the appendix was exposed by lifting the caput coli; the abscess was not in that location; it was found that the appendix had curved inward and the abscess had formed behind the coils of the intestine toward the midline—the umbilical zone. Here you can see the great mesentery covering the abscess as it is exposed. I will open it now with blunt dissection through the mesentery, so as not to injure the mesenteric arteries and veins, as they bleed profusely. A tube is now inserted into the cavity, and from it a large quantity of pus drains freely, and continues to drain, during the completion of the operation, which was accomplished in the usual manner. I put in a few strands of gauze around this tube, and it is the first time that I leave gauze in an abdomen after an operation for appendiceal abscess in over two years. [The patient made an uneventful recovery.]

CHRONIC APPENDICITIS

HISTORY

FEMALE, aged nineteen, single, by occupation a printer. Family history negative. Had ordinary diseases of childhood.

Began to menstruate at fourteen, always irregular, each period lasting seven days; flow moderate, three or four napkins a day. Some pain on first day of each menstruation. Two months ago had slight vaginal discharge. Occasional pain on urination. Last period Saturday, October 7, 1911. Menstruating at present. Obstetrical and venereal history negative.

Present Trouble.—September 21, 1911, patient was seized with pain in epigastrium, which became general in about two hours, and in about five hours localized at McBurney's point. Nausea began almost simultaneously with pain, and in about two hours she began to vomit a greenish-yellow material; the emesis lasted all night. She also felt very feverish during the night. The following day she felt better, but was very tender at McBurney's point. The second day she got out of bed, and on the third day returned to her work. Since this time her left side has felt weak.

At about 9 P. M. on Tuesday, October 3, 1911, patient began to have general epigastric pains, which rapidly increased in severity. Toward morning she felt very much nauseated, and next forenoon vomited once. On Wednesday pain was localized at McBurney's point, where it has remained. She vomited again on Thursday, and had a temperature of 102° to 103° F. She has not vomited since Thursday, October 5th, but has had constant pain at McBurney's point; she is also very tender. Has felt feverish since onset. No lumbar pains and no urinary symptoms. No history of gall-bladder infection, and no tenderness over gall-bladder. No history of pelvic infection. Bowels very constipated.

October 9, 1911: On opening the peritoneum intestines appeared highly inflamed and thickened, and there was evidence of localized peritonitis. In attempting to expose appendix the pus, which had been walled off in right lower quadrant, escaped in large quantities and was of a very foul odor. No attempt was made to remove the appendix, but a large drainage-tube was inserted into the right iliac fossa.

Abdomen closed in usual way. Recovery uneventful.

The patient left the hospital on November 27, 1911, and the incision closed up entirely and ceased to discharge on December 4, 1911. It has not reopened or discharged any pus since that time. She had no pain or symptoms of any kind following the closure of the wound until Thursday, December 21, 1911. The patient states that she had retired for the night and was awakened at 2 A. M. by severe generalized abdominal pains. A few minutes later she was nauseated and vomited a greenish fluid. This was followed immediately by a chill and she felt feverish. She vomited again on Friday, the day following. She has had generalized dull abdominal pains since then, up to the present time, and the abdomen is tender, especially on the right side. Her temperature was 102.6° F. on December 25th. Her temperature on admission to the hospital, December 26th, was 100° F., and the pulse 110.

Examination shows an area of dulness extending from one inch below the incision on the right side down into the flanks on the same side. The superior and inferior margins of dulness reach from the superior and inferior extremities of the cicatrix from the previous incision.

Operation December 26, 1911: Incision was made parallel with the old cicatrix. The peritoneum was much thickened, with many adhesions. Pus was present. A tube was inserted into the walled-off peritoneal cavity—the site of the pus. Another flat drain was placed extraperitoneally, to be removed in forty-eight hours. She had been previously operated for appendicitis, but the appendix was not removed at the time—it was only drained.

February 7, 1912; Patient returned for removal of appendix.

COMMENTS AND OPERATION

The history of this case is instructive in this fact: she had an acute attack of appendicitis on the twenty-first day of September, 1911. She entered the hospital with a large abscess in the right side, and was operated on October 9th, about eighteen days after the onset of the symptoms. She was in bad condition, with a very large and offensive abscess.

No effort is made by us to remove the appendix in these cases under these circumstances unless it comes right into the field, unless it is easily accessible. We do not tear up the adhesions; we do not spread the pus into other areas of the abdomen. It is an entirely different situation where an appendix case comes to us on the second, third, or fourth day. There we have adhesions of the inorganic type—natural agglutinations; while on the eighteenth day we have organic adhesions. When we separate the agglutinations we separate two non-absorbing surfaces. The lymph-spaces are coffer-dammed, and there is not the slightest danger of absorption from the surfaces. When we separate organic adhesions, then we tear up vascular areas—we produce a fresh vaccination zone, and we have all the dangers of infection which we would have if the patient had had no previous inflammatory condition. You see, then, the reason for taking out the appendix on the second or third day in the presence of pus, and also the reason for not taking it out on the twelfth or fifteenth day, because in the first case you only separate agglutinations without courting infection, and in the other you tear up organic adhesions which are easily infected.

The second operation, which was done by Dr. Golden, was a retroperitoneal operation. The abscess was opened and drained without any attempt being made to tear up the adhesions or to find the appendix. She was informed that if she had a second attack of appendicitis she was to come back on the first day of the attack, and we would be able to take out the appendix in the first twenty-four hours. She failed to follow our instructions. She did not come back the second time until she had a large abscess. If we felt she was not going to follow instructions when

here the first time, we would have kept her here, as we have now, and removed her appendix.

We went into this case with the understanding that there are adhesions and the usual difficulties which one expects to encounter, and we are going to try and meet them, at the same time removing the appendix and saving her life. You will remember there was a time in the management of cases of appendicitis when we believed that the right technic was to go in and tear up the adhesions and get out the appendix and make what was known as a one-sitting complete operation. This complete one-sitting operation in 1902 received the greatest indorsement before the meeting of the American Medical Association at Saratoga. At that meeting it was shown that the men who followed the plan of doing a one-sitting operation, or tearing up everything in sight until everything was all cleared, until all possible accumulations were removed and the appendix taken out, had a mortality which was equal to, if not greater than, the mortality of the unoperated cases. At that meeting it was definitely determined that such a line of procedure was not the proper one.

There are adhesions here where the drain was put in the first operation. They are rather firm. Notwithstanding the extensive inflammatory conditions that were present here at the time of the previous operation, absorption has taken place in every position except where there was an organic fixation to the abdominal wall through the drainage-tube, and this is the position of organic fixation to the abdominal wall. It was in the line of the drain. I am freeing that now so as to endeavor to come down into the zone of the appendix, if the appendix is still here and has not sloughed off in either of the attacks. I follow the caput coli to see what is left of the appendix. There is apparently the appendiceal stump. The line of thickening at this point is covered with adhesions. I am freeing them and endeavoring to observe the location of the appendix. There is the stump. Let us see how much of an appendix is left. Here is an appendix, I think. It is embedded or apparently separated from the intestine. It is either the appendix or a tumor. The reason I think it is the appendix is on account of the mesentery,

but it is separated from its organic connection. I will follow that down in order to determine what it is. This girl never could be well because she has occlusion of the appendix just distal to the caput coli. She has a chronic continued infection all the time. She had an obliterating appendicitis, which was so beautifully described by Senn. It is a very pretty picture, showing total obliteration or closure of the appendix where the acute inflammatory attack took place. We treat the mesentery of the appendix in the regular way, just as we would in an intermediate case, or in an acute case, when we are able to bring the appendix up into the field. I am putting in an overstitch to make one continuous circle of peritoneum around that, and with that stitch I ligate the mesentery of the appendix and at the same time cover the whole zone with peritoneum. You can see a chronic exudate around this appendix in the distal portion. While there is some thickening at this point, it is not very marked. That appendix would continue to fill and rupture indefinitely unless taken out. There is no drainage for the infective material in the distal portion of the appendix, and that is why it is so thickened and why it is so distended. Ninety-five per cent. phenol is applied to the stump. A puckering string suture is put in, and the stump is embedded. The first puckering string acts as a Lembert suture; the second is a Lembert suture, but not put in on the puckering string plan.

There has been a chronic inflammatory condition here, with a cobweb exudate that does not adhere and is non-infective. That is the type that we find present where there is a tuberculosis, and we all know that where there is a tuberculosis in the intestine, with this type of exudate, we are in great danger of failing to secure union. There are three means of preventing leakage there: First, the ligature around the appendix; second, the puckering string, which is a Lembert suture, and third, the accurately applied Lembert suture. I am cutting through the scar tissue that resulted from the previous operation through the line of drainage to expose the rectus muscle on the inner side. There is the belly of the rectus muscle. We do this so as to get all the support we possibly can for this line of suture.

We are operating in this case as we would on a ventral hernia. This patient has to earn a livelihood, and we must guard against a hernia at this stage by doing a thorough and systematic operation, and for that reason we are going to make an overlapping flap of the aponeurosis. The aponeurosis is on the outer side of the rectus muscle. The method of closing this incision is different from the ordinary method of closing the abdomen. We are going to make a flap closure, an ideal method for the cure of hernia,—the imbrication method, as used by Andrews in his radical operation for hernia,—and the overlapping flap method as it is used on the same principle by Mayo in his operation for umbilical hernia. The central flap is made the under flap on this occasion. The peritoneum is closed where the flap is. This is the overlapping flap, and it was put in with a continuous stitch. This type of aponeurotic fixation gives the greatest possible security that one can have against a hernia, or we might say it is the best method of preventing a hernia, and it gives the greatest security in the repair of a hernia.

Let us analyze this case from the beginning. Let us see what conclusions we can draw. First, she had the typical classic symptoms of an acute attack of appendicitis; pain, nausea and vomiting, elevation of temperature, local sensitiveness, and the formation of a large phlegmonous abscess. Second, the attack was permitted to continue until the eighteenth day before an operation was performed. As a result of that delay a large phlegmonous abscess formed within the abdomen. On opening the abdomen the condition was such that to remove the appendix at that time would materially have increased the hazard to the patient's life. The first rule of surgical practice is that a patient's life must not be hazarded unnecessarily in any procedure. The matter of whether you do a secondary operation or not is of little value to that patient or to you as compared with increasing the hazard to the patient's life. That is one thing that cannot be restored, and if you take risks, you are certain to pay the price. In these cases we learned very early, when they came to us at the stage we had here, not to do the complete operation. We have never varied from that rule, and we believe that that

has been one of the sources of our success in the management of cases of appendicitis. This woman ran a great risk, and she has paid the price for disobeying our instructions, and for which we are not responsible. When she came the second time the abscess was opened extraperitoneally—the peritoneum was not opened at all. This time we did not let her go home; we kept her here in the hospital while she was convalescing from her attack. She was operated on the twenty-sixth of December, and you can see an illustration of the great absorption of adhesions that can take place in a comparatively short period of time when they are not organic. When they are organic, the absorption does not take place. That means absorption of the adhesions that formed at the entrance of the tube did not take place and never would take place. That is what is known as an organic adhesion, because the peritoneum is destroyed completely through its depth and is not covered again with endothelial cells.

Let the record show that the adhesions had practically all absorbed down to the cobweb formation on the general surface of the peritoneum; that the only organic adhesions remaining were those at the point of entrance of the drainage-tube and down the side of the parietal peritoneum, where the tube drained the depth of the abscess; that the appendix was folded between the caput coli and the ileum; that it was obliterated at its base, and that this obliteration produced the continued retention of pus in the appendix, caused its distention to almost the size of my little finger, and kept the patient in a constant condition of mild sepsis.

A Physician: Will you please tell us what suture material you are using in your figure-of-8 stitch?

Dr. Murphy: This is silkworm-gut dyed black, because it is easier to recognize when we come to remove it. We have used no other material for a great many years, and we rather like it. I do not depend alone for support on my catgut suture. This is an inabsorbable suture of two rows, because the lower loop represents one row, and the outer loop the other, and still neither one is buried—all inabsorbable material.

There was no evidence at any place in the abdomen to show

that the abscess had ruptured into the intestine. If the abscess had ruptured from the appendix into the bowel, we would find organic fixed adhesions between the appendix and the bowel. There was at no time any communication between the intestine and the peri-appendicular abscess.

Let us go back to the consideration of the hazards this patient has had from the fact that her appendix was not removed early in the first attack. We can only repeat now, what we said in 1889, when we wrote our first article on lesions of the appendix, namely, that the time for operation was now! and "now" meant within the first few hours of the attack, and we repeat again the statement we made at Budapest, at the meeting of the International Medical Congress, 1909, that every case of appendicitis that is operated in which pus is found outside of the appendix at the time of operation has, in the presence of that pus, irrefutable evidence that the case was badly managed up to that time. There is no longer any reason for having pus outside of the appendix, as all cases of appendicitis should be operated promptly. The symptoms are so uniform in the acute infection, and the chronic infections never cause abscess to form outside of the appendix; the symptoms are so uniform and regular that "he who runs may read" the diagnosis, and, having read the diagnosis, he has no way of justifying himself for waiting any longer. There was a time when it was justifiable to wait. Now it is not justifiable to wait, because it is the consensus of opinion of those who have had the greatest experience, that you cannot tell, from the course of the symptoms in the first twenty-four hours, what the course is going to be in the second or third twenty-four hours. Inasmuch as you cannot tell what the course is going to be, you are not justified in permitting a case to go into this advanced stage before something radical is done.

In the recognition of appendicitis in the initial stage of the acute attack we always have a regular course. First, pain—*and pain is always first.* I was in consultation in a case this morning in which the question was raised whether or not the last attack the boy had was one of appendicitis. His trouble began with headache. He had nausea and vomiting with his headache

before he had any pain in his abdomen. The pain of appendicitis is the primary symptom. The reason the pain is the primary symptom is: The appendix is made up of three coats. In all but 1 per cent. of the cases infection takes place in the mucosa; at first the mucosa swells and cannot expand on account of the firm inelastic fibrous coat. That causes the pain; that causes the colic, and then, as the mucosa cannot expand, you have exactly the same condition as you would have if a foreign body were obstructing a canal, causing reflex vomiting. There are only one or two efforts at emesis in the primary stage, and rarely ever is there vomiting in the second stage of the disease. Following that is local sensitiveness, which is due to the great distention of the appendix. Following that comes absorption, in a case of infection causing elevation of temperature and causing an increase in the number of leukocytes.

We have had some interesting observations on the relation of temperature to leukocytosis. Occasionally we have a temperature of only 99.6° to 99.8° or 100° F., with a leukocytosis of 15,000, 18,000, and 20,000. In this class of cases, when there is any question at all as to the diagnosis on account of the temperature being 99.6° to 100° F., there is a high leukocytosis, which shows that infection is present. In that way leukocytosis is of value as a diagnostic symptom in probably from 3 to 5 per cent. of the cases; otherwise you can make the diagnosis in the acute attack without a leukocyte count. You base the diagnosis on the symptoms occurring in this order: Nausea and vomiting never precede pain in appendicitis; temperature never precedes pain in appendicitis. *It is pain, nausea and vomiting, local sensitiveness, elevation of temperature, and an increase in the number of leukocytes.* That is the uniform order in which the symptoms occur.

We have had, I think, nine cases of typhoid that came to us with a diagnosis of appendicitis. In the first case we did not know whether the patient had a temperature or not before pain had developed. We operated in this case and found a typhoid ulcer in the appendix. In the second case there was elevation of temperature before the pain for three days. The typhoid

ulcer demonstrated in the appendix accounted for the sudden pain, nausea, vomiting, and local sensitiveness in the third day of the fever. It usually occurs between the third and fourth day. In the third case we had we did not know whether elevation of temperature had been present before the pain, and we operated and found a typhoid ulcer in the appendix. In the remaining six cases we had all that information, and in each case we made a diagnosis of typhoid ulcer in the appendix, did not operate, and each case went through the regular course of typhoid.

You see, therefore, why I am so particular about emphasizing the fact that the temperature does not precede the pain in the ordinary case of appendicitis. It is unnecessary to operate for typhoid appendicitis, because only ½ of 1 per cent. of the cases have a perforation of the ulcer in the appendix—a much smaller percentage than the perforations which take place in the ileum and large intestine. The appendix is not prone to perforate in typhoid ulcer, while the ileum and the large intestine, even down to the sigmoid, are prone to perforate. But you do not need to operate for it, and we do not operate for it.

ANKYLOSIS OF THE KNEE; ARTHROPLASTY; JOINT INFECTIONS

HISTORY

HOUSEWIFE, aged twenty-seven. Entered hospital because of ankylosis of left knee-joint. Mother died of tuberculosis of the lungs while patient was a very young child. She nursed and always lived at home with her mother.

Present Trouble.—Patient lives in Denver, Colorado, and just after Thanksgiving, 1907, she left Colorado, where it was warm and dry, and came to Illinois, where the weather was then very cold and stormy.

December 8, 1907, she was taken ill with a severe attack of tonsillitis, which kept her in bed, with fever, for one week. The day after Christmas, 1907, the patient visited friends all day, making numerous calls, walking far in the snow, getting her feet wet and becoming very tired.

At 2 o'clock in the afternoon, December 27, 1907, the patient experienced a sudden severe pain in the first phalangeal joint of the third finger of the left hand. This joint rapidly became hot, painful, swollen, and tender. One half-hour later she had a severe chill lasting fifteen minutes. She is certain that pain preceded the chill. About 9 o'clock the same night the right shoulder likewise became hot, painful, and tender, and in succession the right ankle, the right wrist, and, last, the left knee, became involved, all within the next twenty-four hours. She was kept in bed for three weeks and had much pain and a high temperature.

All joints became normal rapidly, with the exception of the left knee, which continued to be sore, painful, and swollen. She remembers that the skin over the joint pitted on pressure made by the doctor's fingers. After five weeks the swelling and tender-

ness gradually subsided, but the leg could not be flexed and has remained so ever since.

In June, 1908, leg was flexed under anesthesia, but stiffness still persisted. In July, 1908, another attempt was made to break up the adhesions, and a nine-pound extension was applied, but without results. An opening into joint was never made. It has never been injected.

Urine contains a trace of albumin. Leukocyte count, 8000.

COMMENTS AND OPERATION

This is a typical history in every way. The primary infection took place in her pharynx. The pharyngeal infection was of about two and a half weeks' duration, and then, as the result of additional exposure, there followed a metastatic infection in her joints. She had a chill, followed first by involvement of a number of joints, without any destruction of tissue, and finally concentration of the destructive effect in only one joint, the infection in all the other joints subsiding. That is the rule. That is the typical course of all these cases of arthritis that are called "rheumatism." They are purely cases of metastatic infection, and in the great majority of cases the primary infection can be located elsewhere in the body, most frequently in the mouth, as in this case.

Before we began to study these infections from the standpoint of metastasis, no record was made of the cold, or the sore throat, or the tonsillitis, or the abscessed tooth, dysentery, gastro-intestinal fermentation, etc., that preceded the attacks of arthritis or rheumatism two or three weeks. Why? Because we had failed to connect the primary infection with the secondary arthritis, and the lapse of time between the subsidence of the primary infection and the appearance of the secondary manifestation is usually long in the average infection, except of the streptococcus type, and it led us to overlook the fact that arthritis is a metastatic infection, easily recognizable and easily demonstrable, following a primary infection of some mucous membrane, as a rule. The pharynx is the most common seat of the primary infection, and in the recent litera-

ture you will find that authors have emphasized the importance of disease of the tonsil as a primary focus of infection. The tonsil, in our experience, has been the least common, the pharynx the most common, source of the metastatic infection. Next in order follow the nasopharynx, the accessory nasal sinuses, the teeth and gums; infections of the bronchial mucosa, infections of the lower part of the intestinal tract, such as diarrheas, gall-bladder infection, and so on. It is rather surprising that the profession failed for so many years to connect the secondary infection in the joints with the primary infection of the mucous membranes, when for many years we have recognized the relation between the primary urethral infection and the secondary articular infections of the Neisserian type. The reason we always connected this was that the patient did not get over the urethral discharge before his joints became involved, and he had a painful and sweeping reminder of its presence. The secondary metastatic infection of the joint or joints of the Neisserian type does not take place, as a rule, until the eighteenth day, and about 90 per cent. of the cases occur between the eighteenth and twenty-fourth day after the primary urethral infection. That is not an accident nor a coincidence. It is a habit, a pathologic habit, and it is in consonance with the other types of metastatic infections that occur in joints. An exception to this is the streptococcus infection, where, within forty-eight hours after the appearance of the primary invasion, there occurs the secondary metastatic manifestation in the joint or joints.

I do not think I can give you a better illustration of this than by mentioning a case that occurred in the practice of my former colleague, Dr. Neff, now of Spokane. A patient, after paying his devotions to the goddess of Cloacina, without washing his hands struck a match on the seat of his trousers, and a small sliver from the match penetrated his finger. That evening he had a chill; the next morning he had a phlegmon of his chest; no suppuration, but considerable sensitiveness. In the evening he had another chill, and the next morning the left knee was swollen and distended. He was in charge of Dr. Neff, whose ability cannot be questioned. Dr. Neff immediately incised the phlegmon on the

chest, and made a culture of its contents. He withdrew blood from the arteries, made a culture, and he likewise made a culture of the aspirated fluid from the joint, and all three cultures and smears showed streptococci and colon bacilli. You see where he got the colon bacilli. Inside of forty-eight hours after the onset of the symptoms the joint was filled with fluid containing both microörganisms. It was opened without tube drainage. A number of other joints were similarly opened. He had phlegmonous abscesses elsewhere; all were opened without tube drainage. It is a law, in connection with the infections of joints, never to put in a drain. Open the joint and relieve the tension, but keep out the drain. The man in this case got perfectly well without ankylosis of a single joint. After the aspiration of the joint Dr. Neff put on Buck's extension immediately. Aspiration, no drain, Buck's extension!

It is impossible for me today to go into the details of the method of destruction of joints, but let me point out a few important mile-stones in connection with this subject.

An infected joint is destroyed primarily through the accumulation and the pressure of the products of infection held under tension in the joint, as well as the virulence of the infecting microorganism. Relieve the tension, and nature does the rest. If you have a phlegmon in the thigh, reduce the tension, and the patient experiences relief from pain. The joint has a velvety inner lining of synovial membrane. The outer lining is an inelastic, non-absorbing capsule, capable of sustaining a pressure of from 50 to 120 pounds. It is this capsule that holds the products of the infection in the joint under such colossal pressure. It is a leather bottle, so to speak, of fibrous capsule, and the products of infection cannot break out because of the non-vascularity of the capsule. Finally, the endothelial cells lining the synovial membrane are destroyed, and what results? Ankylosis. The second element in producing ankylosis is the pressing together of the bones of the joint by the contraction of the muscles above and below the joint, which occurs as the result of an endeavor to immobilize the joint. It is that involuntary muscular contraction which most frequently causes ankylosis,

except in the drainage cases. In a large number of these cases we have found that there is one spot of ankylosis, as on each condyle of the knee, for example, or only one spot of ankylosis on one condyle, these being the only points of contact produced by this involuntary muscular contraction.

How can we stop the destructive effect of these two factors in the production of ankylosis? One, the increased interarticular pressure caused by the accumulated products of the infection, and the other, the muscular contraction. The latter is easily stopped in all large joints by the application of Buck's extension. All that needs to be done is to relieve the pain, and that can be effected by applying a Buck's extension. It is much better than to give ⅓ or ½ grain of morphin. The patient will not allow you to take off the weight, because the moment you do so the joint becomes painful. The pain is also an indication of the destruction that is being produced in the joint by pressure. Remove the pressure, and you relieve the pain and lessen the amount of destruction.

You can stop the destructive action of the accumulated products of infection in the joint in two ways—either by incision into the capsule and permitting the pus or the purulent fluid to flow out, or by aspiration and injection. By aspirating you remove all the fluid that will come out, and then inject into the joint a fluid that will render the contents of the joint sterile, or make the fluid in the joint a bad culture-medium for every type of microörganism that is likely to get into the joint.

The fluid we inject into the joint is a 2 per cent. solution of formalin in glycerin. Inject into the joint 10 or 15 c.c. of this solution that is mixed at least twenty-four hours before it is used, because it requires twenty-four hours for the formalin to dissolve with the glycerin. That renders the joint a bad culture-ground. Reaspirate the joint from day to day, or every second day, keeping the tension off, and in a little while the synovial membrane becomes coffer-dammed and absorption of septic products ceases. The formalin and glycerin solution does more than to render the joint fluid sterile, or to act as an antiseptic. It causes an enormous outpouring of the polymorphonuclear or

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polynuclear leukocytes. They are the real phagocytes. They have not only the power of grasping and holding the microörganisms, but they contain trypsin. That trypsin is freed on their death. It is a ferment. It converts albuminoids into peptones. This phagocytosis and digestion rapidly render the joint sterile and thus overcome the infection. Prevent ankylosis by extension, and inhibit destruction of tissue by aspiration.

Every acute infection of a joint, call it "rheumatism," arthritis, or whatever you wish, should be treated as you would treat an infection in any other tissue in the body, with the one exception—that you cannot put in a drain and allow the air to come in contact with the synovial membrane. If you do, you violate the first law to be observed in connection with synovial or serous membranes in every position in the body, namely, that exposing them to the air for a considerable length of time will cause destruction of the endothelial cells and lead to the formation of adhesions and ankylosis. These are all simple things, but every one is vital to the final result, and every one can be utilized to avoid the awful catastrophes that occur to joints as a result of infection.

This patient is one of the best cases to illustrate this. She has little or no deformity. She has practically a straight limb. The skiagram shows that there are only two points of ankylosis absolutely fixing the joint. This accentuates what I said, namely, that if she had had Buck's extension for the first two weeks of joint inflammation, she would not have had a bony ankylosis at all.

There is fixation of the patella. I do not think that it is a bony ankylosis—merely a rather firm fibrous fixation. I hope it is not bony, as it will save me turning the patella turtle. The worst trouble we have had in any of these cases has been a necrosis of the external skin-flaps, and it has been the most annoying trouble we have had. We will now make a U-shaped incision to expose the joint, commencing two inches above and one inch to the outer side of the upper end of the patella, passing downward to about the level of the tibial tubercle, and then across the tibia toward the inner side, with the convexity of the incision down-

ward, passing upward to the inner side of the patella to a point opposite the starting-point. This incision extends through the skin and fascia down to the capsule of the joint. Next I will divide the capsule lateral to the patella to get my interposing flaps, which have to go in between the joint surfaces subsequently. One flap will cover the outer condyle, the other will cover the inner condyle. A U-shaped incision is made on both sides of the knee, the base of the flap being directed downward and on a level with the articular surface of the tibia. The next thing to do is to loosen this patella. There is only a fibrous fixation. That is a great relief.

In this case I will not need the long tissue flap, nor will I need to rotate the patella. Next, we will find the line of demarcation between the femur and the tibia. I am following that line around so as not to disturb it any more than is necessary. I shall free this patella from its fibrous attachment to the femur.

Now we are in the joint zone. The posterior portion of the capsule has not been touched. It is intact. The lateral portions have been converted into the interposing flaps. We are ready to make the new bone conformation for the joint. This conformation of the ends of the bones is made with straight and curved chisels of various sizes. Sufficient allowance must be made for the interposing flaps, and the leg must be plumb. We keep the intercondyloid ridge on the tibia intact. That is essential to the final result. If you do not keep this elevation intact, you will have a slipping of the femur from side to side. You see how much space I have here between the femur and tibia—⅓ of an inch. We are getting ready now to put in the interposing flaps. The flap which was taken off the inner side I will now suture to the stump of the crucial ligament behind, so that it covers all the articular surfaces of the internal condyle of the femur and the inner tuberosity of the tibia. For suture material we use phosphor-bronze wire, 12 strand, not chromicized gut. It is non-absorbable, flexible, and is not irritating. The outer flap will cover the external condyle. It is drawn in at a different plane, because it goes back to the internal condyle. It overlaps the edge of the internal flap. I will fix this in position so as to cover the margin of the tibia and

catch the periosteum of the tibia. I am putting in just one understitch, so as to be sure and have this flap overlap in the middle, because there is where you have the greatest danger now.

We are endeavoring to do here just what nature does in causing non-union of a fracture—interposing fascia and fat between the bone-ends. We know that in ununited fractures this is the cause of most cases of failure of union. Here, that is just what we want.

The next step is to place a portion of this flap which I have brought from the vastus internus between the femur and the patella to prevent the recurrence of fibrous adhesions between these two bones. We could not get bony union because we do not have a cartilaginous surface, but we would get a fibrous fixation which is equivalent to bony union as far as the result is concerned. The remnant of the vastus internus I am fixing to the patella, so that it will not subluxate outward. I have to turn the patella bottom side up, and in doing that we have the disadvantage of traction. So much for the upper stability of the patella.

For the lower stability of the patella, here is the tendon patellæ and there is the fascia. We bring that back and unite it, so that it cannot slip to the outer side. The subsequent luxation of the patella is always to the outer side. One rarely ever has to put on a ligature to stop bleeding. All oozing can be stopped by compression. I will secure the sides of the ligamentum patellæ, so that it will not tend to inversion or eversion subsequently, but will stay in the exact relationship to the tibia in which it was before.

This is the same plan we have followed in all our cases, with the exception of the management of the patella. We manage the patella in four different ways, depending on the case:

1. By using an interposing flap from the vastus externus and vastus internus to prevent reconsolidation of the under surface of the patella with the femur.

2. Splitting it into two parts from above downward, and then turning the upper half under the lower half, so that the smooth aponeurotic surface came next to the femur, thus making it impossible for bony union to take place.

3. Freeing the vastus externus and internus attachments for a distance of two inches above and below the patella; next dislocating it from side to side during the operation; and when the limb is straightened out and the interposing flap is in position, a 180 degree rotation of the patella is made, so that the upper surface of the patella now becomes its articular surface. The vastus externus and internus are now sutured to the opposite sides from which they were freed, to prevent luxation of the patella and the return to its former position.

4. Covering the under surface of the patella and the entire articular surface of the femur with a fascial graft taken from and detached from the trochanteric zone of the fascia lata, without rotation of the patella.

I have had good results with all these methods. The rotation, however, is the simplest one, and after operation gives an additional leverage to the quadriceps tendon. It has some disadvantages, as it taxes the vitality of the skin-flaps.

There is a little oozing from the surface of the bone. I am taking the tension off of the line of skin suture and putting it on the deeper structures. The tendency of the flap is to pull outward. I have put that away back so as not to interfere with the circulation at the edge. We close the wound in the usual way, using catgut for the deep sutures and horsehair to approximate the skin incision. Subiodid of bismuth powder is dusted on the wound, and this is then sealed with gauze saturated with collodion. A four-fifths plaster-of-Paris cast is applied, extending from near the groin to below the ankle. There is practically no tension at all on the line of suture. Remember, I fear necrosis of this flap more than anything else in connection with this operation.

Let us recapitulate: In this case the ankylosis was of the condyles close to the middle line, near the intercondyloid ridge of the tibia, and the patella was merely fixed by fibrous adhesions, as interpreted by us before operation. Both of these suppositions were sustained by the findings at the operation. This is rather extensive work, and involves a certain hazard in connection with infection. That is the greatest danger of all these procedures,

because you have to change your position often, and because you have so many instruments, sponges, etc., in the field.

We have had one recurrence of ankylosis of the fibrous type in an elbow. The patient left the hospital shortly after getting out of bed. She would not submit to the massage and exercise that were ordered after the operation. The result was that the ankylosis recurred and the arm is as stiff now as it was before the operation. We have not had a single recurrence of a bony ankylosis of the knee. We have had one infection, namely, osteomyelitis of the femur, following an operation with some necrosis. We had an infection in a case of hip ankylosis where the patient, a morphin habitué, was deprived of the morphin, and on the twelfth day after the operation he tore off the dressing from the wound; infection set in, and he died three weeks afterward from infection. It was the second operation on the same patient. We had operated previously on the other hip and secured a splendid result.

The best joints in which to do the operation are, first, the hip; second, the elbow; third, the knee. The knee requires the greatest mechanical adjustment of any of the joints. Unless the limb is perfectly plumb after the operation, the knee will be drawn outward or inward. In chiseling the ends of the femur and tibia so as to restore their normal anatomic conformation I endeavored to get it exactly plumb, so that the pressure would fall evenly on the external and internal tuberosities of the tibia and on the external and internal condyles of the femur. In this way we hope to be able to have this patient carry her weight without the slightest deviation of the plumb-line of the leg. It was not so difficult to do that in this case as it has been in many of the other cases, because here we had the limb plumb to start with. In the great bulk of our cases we have had great deformity associated with the ankylosis. In some of them we have had the angle so acute that the heel practically touched the buttocks. The angle at which the ankylosis takes place very materially increases or diminishes the risk which one assumes in doing the work. It also adds to the mechanical difficulty of the operation. The straight limbs afford the best conditions for securing good mechanical results.

After the skin wound has been carefully closed with horsehair, the site of operation is dusted freely with subiodid of bismuth. It is a desiccating powder, rather than an antiseptic. We never drain in these cases. A moist carbolic gauze dressing is then applied. The reason we use carbolic gauze dressing and not the ordinary dry gauze is that there is always some bloody oozing into the gauze, and in the dry gauze the blood decomposes. If it oozes into this carbolic gauze dressing, it does not decompose. It remains sterile; no saprophytes can live in it. It is the saprophytes which we fear. There is only the slightest pressure over the flap. There is no pressure at all over the patella. That is where the greatest danger is. We shall put on a Buck's extension with a ten-pound weight. The leg is placed in a wire cage and elevated from 14 to 16 inches, otherwise the foot will swell. We use a double-inclined plane. The heel will not be hazarded in this case.

In doing this work you are on edge all the time. Every single step must fit to get a result. It is the little things that count in all the technic of joint work. They must be done with precision.

Here is a patient who had an ankylosis of both hips. The first operation was done seven or eight months ago. The second operation was performed two weeks ago. He also had an ankylosis of his spine extending to his second cervical vertebra as the result of so-called rheumatism. Both hips were fixed. He could not sit down for nine years, because both of his hips were ankylosed. Now he has a movable joint. He can bring his leg up to a right angle with his body, swing it, abduct it and adduct it. He also has a considerable degree of rotation. Before we operated on the second leg he could bring the leg operated on first almost to or better than right angles, showing what can be done in these cases.

Here are some illustrations of another case. (See Vol. I, No. 3, p. 301.) This was a most difficult case from a mechanical standpoint. The ankylosis of the knee began when the patient was seven years of age—some thirty years ago. We encountered enormous difficulty in getting the leg straight. It was bent so much that with the thumb on the buttock and the fingers spread, the little finger touched the heel. The femur was bound. We turned the

patella turtle in this case. We had to remove an inch and a half of the femur in order to get a straight leg, and still we secured a good anatomic conformation. In straightening the leg we had to stretch the nerve and blood-vessels so much that we feared gangrene would occur. She had complete paralysis of the foot from stretching of the nerve. That paralysis is rapidly disappearing. She was operated on fourteen weeks ago. There is perfect freedom of motion of the patella. We turned the patella over, so as to put the prepatellar bursa underneath to form the lining of the joint. She has a little ulcer on the heel, because she had no sensation, and the pressure on the heel caused the ulceration. The flexors are already acting; the extensors will be acting shortly.

Here is a case (see Vol. I, No. 3, p. 303) in which we did another type of operation for ankylosis. We had to take off the head of the tibia down to about the level of the fibula. The patient had been operated on before elsewhere, and there was so much cicatricial tissue around and about the knee that we could not secure the customary interposing flaps. We removed the trochanteric fascia lata and used it as the interposing flap. We have succeeded in getting splendid separation of the bone, and we are going to get an excellent result. That is the final thing I planned to do nine years ago. I began doing flap operations of one type after another. I predicted then that the time would come when I would be able to take a flap from some other portion of the body and place it between the bones to serve as the interposing flap, but I had never had a case in which I was forced to do it, and inasmuch as I was getting good results with the usual method of making these flaps, I did not feel justified to try a new method. She had no patella, the patella being merged into the femur. I chiseled that out and made a new patella. There was healing by primary union. She has some function, but not as good as it would be if she could be prevailed on to carry out the passive motion and exercises prescribed in these cases. The anatomic result is excellent.

The next case was one of very bad Pott's fracture with a posterior luxation of the astragalus. (See Vol. I, No. 3, p. 451.) The operation was done several weeks ago. We brought the

astragalus a little anterior. We refractured the internal malleolus because it had united to the tibia in a position which caused the anterior displacement of the bones of the leg. Then we nailed both malleoli into their normal positions with wire nails. The result is excellent. We have bony union, and all the deformity has disappeared. The leg is straight, the foot can be placed squarely on the ground, so that she has a perfectly normal leg. Motion is already very good.

Here is another illustration of a Pott's fracture complicated by a spirochetic infection. (See Vol. I, No. 2, p. 151.) There was inversion of the foot with absorption of bone. We took out all the dead bone, freshened the surfaces, and drove in a nail from below through the astragalus and straight up into the tibia. We hope to get bony ankylosis in a Charcot joint. He has a serviceable ankle. We also drove a nail through the tibia and fibula as a lateral support and splint. We fractured the fibula above the joint. He will have a bony ankylosis in his ankle, so that he can use it and make his living.

The next case is one of a fracture through the anatomic neck of the humerus, with dislocation of the head of the bone downward into the subglenoid space. The patient came to us about three months after the accident. We liberated the head, took it out on the table, freshened the fractured surface, replaced it in the glenoid cavity, and nailed it in place with a wire nail driven in from the outer aspect of the humerus at the greater tuberosity. There is perfect regeneration of the head of the bone, with return of normal function in the joint. We secured the same result in a hip case. The fragment, consisting of the head of the femur which had been broken off from the bone at the anatomic neck, was rotated and had strangulated the ligamentum teres. The patient came in seven weeks after the accident. The head was dead but aseptic. We did not take it out; we turned it around and nailed it back in place. The head of the bone has been completely regenerated. It is now about three years after the operation. Function is normal in every respect. There is a shortening of less than half an inch.

Bone always regenerates, no matter how long a transplant

you put in, if you contact it at both ends with living osteogenetic elements. The transplant acts as a support for the Haversian vessels going from the living bone into the Haversian canals of the dead bone, carrying the osteoblasts and osteoclasts and filling up all the dead portion. If you take away all the fragments of dead bone, the osteoblasts will make new bone of identically the same shape and size as the normal bone. The only rôle the transplant plays is a mechanical one, that of carrying the Haversian vessels.

This is a case of ununited fracture of the humerus. (See Vol. I, No. 2, p. 193.) A Lane plate was used to maintain perfect approximation and still there was failure of union. We took out the Lane plate, waited until healing had taken place, and then put in a 4½-inch splint taken from his tibia. The skiagram shows that bone regeneration is progressing rapidly.

We recognize five indications for transplantation: First, in ununited fractures; we never adjust ununited fractures with plates or wire. We put in an interosseous splint. Second, for the removal of infective diseases of bone, as in osteomyelitis, where there is much necrosis of a portion of bone. Take out the dead bone and allow healing to take place. Third, for the removal of non-malignant neoplasms, that is, of the fibrous and cystic variety of bone disease. Fourth, for the filling up of aplasic conditions where the bones fail to develop as they do in the arm and tibia. Fifth, for malignant diseases of bone, as sarcoma. These are the conditions in which regeneration of bone can be effected with mathematical certainty if you secure and maintain perfect asepsis. We have not had a single case of failure from sepsis; not one. In arthroplasty, if, following a transplantation of bone, sepsis should occur, you must take out the transplant, do the work over again, and do it better than you did the first time.

I demonstrate these cases so that you may note their progress from week to week, and in that way get a better idea of what is to be expected from such cases.

ANGIOPHLEBITIS OF LEG AND THIGH—OLD MUSCULAR HEMANGIOMA

HISTORY

Mr. M., clerk, aged thirty-seven, entered hospital because of a deformed foot and leg. Had malaria of the tertian type at twelve. Recovery excellent. Never had rheumatism or any soreness or disease in any other part of body. Never used alcohol in any form or any other drug. Neisserian infection eight years ago. Recovery apparently absolute. No joint or bone pains afterward. Denies lues in family or self. No tuberculosis or malignancy.

Present Trouble.—At the age of seven he noticed that he could not flex the right foot dorsally, though he could run and play without limping. He wore out the sole of the right shoe rapidly on the inner side. When ten years old, he first noticed that his right leg would not touch the floor when standing erect. Right leg was then slightly smaller in circumference, and his mother noticed that the outer side of the right calf presented a sunken appearance. The deformity seemed to develop gradually, the foot and leg drawing up more each year. Ten or twelve years ago the hamstring muscles began to contract, pulling leg upward until it cannot now be straightened out at all. The fibular region has been sunken for the past ten or fifteen years. Leg never has been injured in any way, nor has it discharged pus, nor has there been a sinus leading into it. The veins on the dorsum of the foot are distended and calcified. A very slight tenderness has existed in the limb during his whole life. Foot has never felt cold and never has been the seat of edema or swelling.

Examination by Intern.—No evidence of rickets found. No asymmetry of head or chest, but mandible is unusually weak and

prominent at posterior portion near the angles. Talipes equinus, with a little varus, of right foot. Contour of the fibular region not maintained; presents sunken appearance, with thickening and bony feeling deep under skin at posterior and lower portion. Both hamstrings are contracted, and on deep palpation posterior thigh feels slightly indurated in places. Lungs and heart are apparently normal. Urine normal.

Further Examination by Dr. Murphy.—A venous angioma of right foot is present. It seems to be typical of the changes found in all these cases. Atrophy of both muscles and bones has occurred. Venous angioma exists in both gastrocnemii and in the soleus muscle, but mostly the gastrocnemii. It extends upward from five inches above attachment of the tendo Achillis. It is hard, woody, and pains on deep pressure. Posterior tibial artery is plainly visible through the skin behind the internal malleolus. It looks calcareous, but this is not confirmed by palpation. Patient was informed that operation would put excessive tension on vessels of popliteal space and might cause gangrene. The two internal hamstrings will need to be divided. Three tendoplasties will have to be performed on the inner side of the foot. Adductor group of muscles is very materially shortened.

COMMENTS AND OPERATION

We have had cases similar to this at this clinic, but not in the stage in which this man presents himself to us. The description of the veins, as it was read by the intern, is not correct. He apparently had an enlargement of the veins on the anterior surface of the foot. That is my recollection. From the time he began to walk he always wore the sole of the shoe out near his toes, and it was difficult for him to get his heel down. The key to the situation is the fact that he had an enlargement of the veins of the foot and toes from birth, and that is what led to the diagnosis. He has now no special enlargement, but still there is some enlargement of these veins, and when you palpate his toes, you will find he has a diffuse venous angioma of the lower half of the great toe. The muscles are all absorbed, the vitality

Fig. 101.—Skiagram showing calcareous deposits in small vessels of foot.

Fig. 102.—Skiagram showing vessels of upper two-thirds of thigh free from deposits.

of the tissue is greatly impaired, and it feels like bone surrounded by skin with a soft cushion of fluid beneath.

When you palpate two inches above the tubercle of the os calcis, you will find there is a solid mass running all the way up to his popliteal space, and extending through the popliteal space and up four or five inches in the fold of his thigh. It is hard, even bony in many places, and always sensitive to pressure. The talipes equinovarus—all the deformity—is due to the continued and increasing constriction of his gastrocnemius and soleus muscles.

When you feel the muscle, you ask yourselves the question, Is this a case of myositis ossificans or of some other lesion? It feels as hard as bone. The key to the situation, however, is the fact that on the foot there is still present a venous angioma. He had a venous angioma originally involving his gastrocnemius muscle, the popliteal space, and the lower third of the thigh. That venous angioma had a phlebitis start in it, and then he had a thrombophlebitis, with the formation of phleboliths, all through that muscle. Then came the gradual contraction of the muscle. The skiagram shows the condition beautifully. There is periphlebitis extending down to the external malleolus. There are two inches in which there is no deposit, commencing on the outer side, over the articulation of the fibula, then extending up from the attachment of the biceps to the fibula, then passing up and appearing again on the inner side. There is an active venous angioma, without thrombosis, in the popliteal space.

We want to try and get the foot straight. I want to see how straight the knee comes under traction. I have to let the leg move back as soon as I straighten it, because the heads of the gastrocnemius muscles are attached to the posterior outer aspect of the external and internal condyles of the femur, above the level of their articulation. Stretching that by any means except by operation would be ineffectual. When we free this, it is a question how much trouble we will have with the capsule of the joint.

This foot has been in this position since birth, I understand, so that we are likely to have trouble in lengthening the posterior capsule of the joint, and we may have to do a capsulotomy on it.

There is danger of gangrene occurring in this leg in two places, and this danger has been distinctly stated to the patient. The artery is diseased, thickened, and thrombosed, so that we have a fear of fracturing the posterior tibial artery when we bring the foot forward, and that may cause gangrene. When we make extension of the leg, we may produce sufficient compression on the popliteal artery to interfere with the circulation in the popliteal space; it may be compressed by a phlebolith, and then gangrene of the leg would occur. This man had to be prepared for the dangers of this operation, and he had to accept them. A positive statement was given him before we decided to operate. That is the proper way to deal with these cases. We cannot assume the responsibility for him. He must know the danger to which he is exposed.

The tendon of the gastrocnemius is displaced inward by the phlebolith. We are not going to cut out the phlebolith, but we will elongate the tendon. The superficial veins we must expect to be enlarged here. Why? Because the deep ones are occluded.

We will make the first incision longitudinally over the tendo Achillis, which we will elongate in order to put his foot into a normal position. We draw the foot away up and divide the tendo Achillis by a double L (⅂) shaped incision. We need an elongation of nearly three inches. Therefore we cut across the tendon half way at one point, from without inward, then half way again, but from within outward, about an inch and a half below the first incision, and unite the two transverse incisions by a longitudinal incision through the center of the tendon. You can see that is just a little over an inch and a half division. By letting these two pieces slide upward and downward, respectively, we get the full three inches of elongation. I will put in two over-stitches with the same thread, merely to keep the tendon ends in apposition. Remember, the tendo Achillis spans a greater distance then any other tissue in the body except the trifacial nerve. This stitch merely keeps the ends in apposition. There is no tension on them. The foot now comes up without a particle of trouble, so that we feel perfectly comfortable about this condition.

Fig. 103.—Skiagram showing calcareous deposits in veins of leg.

Fig. 104.—Skiagram showing calcareous deposits in veins.

The reason I will not immediately put the foot in a cast in that position is for fear of the posterior tibial artery, which is calcareous, as shown in the skiagram. We may stretch or injure it, but if we will let that stay loose—we will get all the collateral circulation we need to prevent gangrene. Therefore there will be the least likelihood of necrosis of the foot from interference with the circulation in the posterior tibial artery.

In the restoration of these limbs to position they do not come around as readily as this foot did, and the reason that they do not do so is on account of the contraction of the posterior portion of the annular ligament of the ankle-joint. When this is hard to stretch or when it does not tear readily, then I cut clear across it with the tenotome, in the direction of the joint, so that a little pressure fractures it. It is important to do that if you have great pressure, otherwise you are likely to traumatize the ankle-joint in bringing the foot around, but there is no injury done by the scalpel or tenotome except the division of the capsule. You know you are anterior to the posterior tibial artery and you have nothing to fear from that source. The skin wound will be closed with horsehair.

The next thing is to straighten the knee. I let the gastrocnemius give first, because that has to give below and is not going to be cut above. The gastrocnemius can retract to a greater degree than any of the long muscles except the sartorius. When I bend the knee so as to straighten the leg and put tension on the head of the gastrocnemius, it can give from below. That is why I did the tendoplasty on the tendo Achillis first.

Next comes the division of the gracilis, sartorius, semimembranosus, and semitendinosus, the inner hamstring muscles. I am making the longitudinal incision for exposure of the hamstring a little to the outer side, so as not to get it directly into the middle of the popliteal space. Here is one of these angiomatous vessels right in the popliteal space. The tendon is now exposed. This elongation I will make on the same plan as I made the elongation below. Remember that these muscles combine and are attached to the tuberosity of the ischium, so you have to free them as much as you possibly can in order to

give the greatest possibility of extending them. I know that this is the tendon by its glistening appearance; in that respect it differs from the nerve, but I will not cut that tendon until I have made the test with the excitor to show that it is not the nerve. I have split the tendon longitudinally, but I have not cut it. Here comes the short muscular tendon where the muscle extends clear down to and forms a cushion on the inner side of the knee, almost to the attachment of the tibia. This muscle must be divided with the tendinous portion that is beneath, from which it derives its name. We must divide that muscle and suture it too. I am making a four-inch elongation. We have the four muscles—the semimembranosus, the semitendinosus, sartorius, and gracilis—coming down here in a bunch. Let us go in and get the nerve, which is still farther back. It shows exactly where you are and you know just what you are doing. While vision is sufficient in this case to guide us right, we cannot overlook the help which the excitor gives us. We will now cut the tendon across above and below at each end of the long incision and suture it in a different manner from what we did the tendo Achillis. The ends will be overlapped just enough to hold them in apposition. That is all. Nature does the rest—fills in the defect and spans the distance, but not so long a distance as it does in the tendo Achillis.

There is the neoplasm or the thrombosed angioma occupying quite a large space. I will extend the leg, but not put on fixation apparatus for fear of compressing the artery. I am going to put on a Buck's extension, which will finally bring the leg into position, but with the least hazard to the patient.

The outer hamstring will be elongated in the same manner. It consists only of the tendon of the biceps femoris.

Just three months ago we escaped having a necrosis of the leg by the closest kind of margin. It was a case of ankylosis of the knee, with the heel almost touching the buttocks. It had been in that condition for thirty years. We elongated the hamstrings and did an arthroplasty on the knee, straightened the leg, but not to complete extension, and put on a cast. The next day the foot was edematous, and the second day it was still more

so. The first day it was cyanotic, but the second day it had a little more of a pink color, and the first day we had motion in the muscles of the toes. The third day motion ceased, showing that the axons of the popliteal nerves had been traumatized by the severe extension. About the third day the patient began to improve, and went on to a complete recovery, with only a slight necrosis on the heel. That, I think, was due to pressure from without. Having lost sensation in the leg, she did not know the heel was being pressed on. The leg was saved, however. She has now a straight limb with a movable knee-joint after thirty years of ankylosis. Motion in the knee is very good.

The greatest flexing force of the leg on the thigh is the biceps, and the biceps, you know, is attached to the upper end of the fibula and along its posterior margin. There it stands out, as you see, conspicuously and strong. Remember, the greatest danger in dealing with the biceps lies in interfering with the external popliteal nerve, which comes right along the inner margin of the muscle, turns over the groove on the head of the fibula, and supplies all the extensors of the foot, so that the popliteal nerve has to be guarded carefully in doing this tendoplasty on the tendon of the biceps. If you cut the external popliteal nerve, you paralyze permanently all the extensors of the foot, which the patient needs so much. He needs his extensors more than he does his flexors. There is the angioma. The nerve is right beside it; in fact, the angioma involves the nerve. It is in the nerve-sheath. That is going to come around beautifully with that slight extension. We will bring the leg almost straight, then we will let it relax again so as to protect the circulation for the next two or three days, then we will put on a weight and pulley to pull the leg straight. The same will be done with the foot. It will be kept up only periodically to start with. We put nothing on the foot now but a dressing.

Let the records show that after exposing the tendon of the biceps the external popliteal nerve was exposed and excited with the faradic current, showing it was not injured. The varix was easily demonstrable in the popliteal space—some portions of it hard, some soft and very vascular. This [bending the limb

gradually] is one of the nice things which we learned from Dr. Lorenz, doing it slowly and repeatedly and a little farther each time, thus avoiding the danger of creating a fracture and causing considerable trouble.

VISITING SURGEON: Will these deposits in the veins disappear?

DR. MURPHY: The deposits will never disappear. They are typical phleboliths. Phleboliths do not disappear. They are permanent. They are mistaken sometimes for ureteral calculi in making skiagrams of the pelvis.

A dressing will now be applied. The foot is allowed to assume any position. We do not put the slightest pressure anywhere, because we do not know how much these arteries have suffered by that extension.

Note.—Three months after the operation the patient stated in a letter that his leg is straight, that he has full motion, and walks without any support whatever. He does not even limp.

HYPERTROPHY OF THE PROSTATE

HISTORY

Mr. W., aged sixty, admitted to Mercy Hospital April 4, 1912. Came to hospital because of frequent and painful urination and a burning sensation during the act. Family history negative. Contracted lues thirty years ago, followed in four or five weeks by a skin eruption. Hair did not fall out. Had mercurial treatment for one year, and no manifestations of disease since. Had gonorrhea twice, with a urethral stricture following the second attack. Had typhoid at ten, with good recovery. General health during adult life has been good.

Present Trouble.—About twenty years ago, following the repeated use of sounds for the dilatation of a urethral stricture, patient had to get up once or twice at night to urinate. The act was attended by slight burning pain. The pain disappeared in three months, but a slight frequency in urination persisted until about eighteen months ago, when it became worse and patient had to urinate four or five times every night. At first there was no pain during the act, but later, preceding each urination, he had rather severe pain, was slow in starting the stream, and at the end of the act still had a desire to urinate. This condition continued for six months, when retention set in (which was one year ago). A doctor was consulted, and he catheterized the patient. Catheterization was continued twice daily for two weeks, the bladder being irrigated after each evacuation. Following this the patient was able to urinate small quantities at a time, with slight pain, but felt that he never could empty the bladder. Two weeks ago patient again had retention, preceded for ten days by very severe burning pain. For the past two weeks he had to be catheterized twice daily. Has not noticed blood in his urine for the past twenty years. He did, however, see blood

after sounds were passed. Patient is extremely nervous. Urine contains some blood and a little albumin.

COMMENTS AND OPERATION

This is an interesting case, and it has peculiarities about it which attract attention. This man has a hard, nodular, woody, and indurated prostate, extending out to the extreme margin of the pelvis. It is a little suspicious of malignancy. It is not very movable around the neck of the bladder. The urine was said to contain some pus and some albumin. He has no evidence of a renal lesion. He is sixty years of age, but does not look over fifty. He is well preserved. He is not the type of man in whom we would look for an enlarged prostate as a senile manifestation, and this is not a senile prostate. He has enlargement of the prostate from an entirely different condition.

If you analyze the lesions of the prostate that bring the patient to you for an operation you have, first, the cases of infection, not because they are more frequent, but because they occur earlier in life. Second, the hyperplasias of the prostate, the senile hypertrophy, as a rule a fibroma, and this condition occasionally undergoes cystic degeneration, causing the formation of small cysts. Third, malignant lesions in the prostate, namely, carcinoma and sarcoma, the former being by far the more common of the two.

Subdividing these further there are, first, the superficial infections of the prostate that occur early in life as the result of a gonorrhea. They occasionally produce an abscess in the prostate, swelling of the perineum, chills, and fever. These patients are intensely sick. Second, there is the tuberculous variety, the disease invading the crypts, slowly infiltrating the gland, and finally appearing in the capsule as a hard mass, with healthy prostatic tissue all around it.

Then there are the general infections of the prostate, not simply an abscess, but a diffuse chronic infection of the gland the result of a chronic Neisserian infection or repeated catheterization. There are diffuse infiltration, diffuse enlargement, caused by an increase in the connective tissue, and hyperplasia

of the glandular structures, the latter not being so marked as the former. That extends and usually involves both lobes. In that type of prostate there occur a slow infiltration and fixation of the capsule to the gland tissue, which make it so difficult to enucleate the gland. If you get the case in the early stages of the process, during the infiltration stage of the disease, you can easily enucleate the gland, as you can all freshly infected connective tissue, but when the connective tissue of the capsule has extended down into the gland between the lobules, so that there is formed a dense, cicatricial, and infiltrated mass of tissue, it is very difficult to enucleate the gland. You have to tear it out. It does not come out from the capsule like the ordinary prostate does.

The ordinary hypertrophies of the prostate involving the three lobes are divided, first, into the small fibrocystic prostate; second, the intermediate sized prostate, with irregularities in its lobes, and, third, the very large hyperplastic prostate.

A prostate produces obstruction not in proportion to its size, but in proportion to its interference with the outflow of urine from the bladder by pressure or contraction. A little prostate not larger than two almonds, placed side by side, with an irregular fibromatous mass projecting into the urethra, will often produce more obstruction than a prostate which is much larger. Therefore, when you examine a prostate, the size of the gland is important only if it produces any obstruction to the outflow of urine. There may be a torsion of the urethra from the irregular growth or hyperplasia with contraction of the gland. These are the dangerous ones that are kept under treatment for a long time. There may be a little bit of a lobe between two big ones, but, as I said, the size is not the essential element in all instances.

Interference with the prostate is indicated when it commences to lead to what is known as catheter life. You do not interfere with the prostate solely because it is enlarged, but when the patient reaches the stage when he has to use a catheter constantly to withdraw his urine, we should operate. When a patient has a primary attack, the prostatic obstruction may be caused by a cold.

A metal sound is passed through the urethra into the bladder, to act as a guide during the enucleation of the gland. I will cut through the abdominal wall just above the symphysis, and as soon as I get through the skin and deep fascia, I divide the heads of the recti on both sides, so as to get into the space of Retzius. Then I displace the heads of the recti upward and split the fibers of the aponeurosis in the midline. The peritoneum is next incised, and we see the wall of the bladder. I am pushing the peritoneum well back so as to bring the bladder into the field. Next, we insert into the bladder what we call the anchor stitches, so that the bladder cannot get away from us at any time during the procedure. If one uses traction on the bladder, he does not let the bladder get away so that he has to open up the space of Retzius to locate it again. The bladder will now be opened between these traction sutures, and then we will let the urine flow out.

Passing my finger down into the bladder I can easily palpate the prostate. It is, as I said, hard and infiltrated, mostly on the right side, crowding into the urethra. The slight enlargement we described before operating is there and of the type we pictured. There is practically no enlargement at all on the left side.

Let the record show that the right lobe is enlarged and firm, and that there is a fixation at that position. It is very difficult to remove it. The small gland is much the more difficult to free. The finger only is used to shell out the gland, enucleating one lobe at a time. The sound is an excellent guide in doing this work. There is not much bleeding. Pads wrung out of hot water easily control it. The key to all this work, which was brought out first by Freyer, but was not accentuated by him, has not been fully appreciated by the profession.

Having enucleated the gland, we will put a large-sized drainage-tube into the bladder through the abdominal wound, remove the traction sutures, and stitch the bladder around the tube. A rubber catheter is passed into the bladder through the urethra after the sound has been withdrawn. This catheter is fastened to the prepuce by means of a silkworm-gut suture.

We then close the peritoneal wound in the usual manner, suture the recti muscles back into place with catgut, close the

sheath, and then sew up the abdominal wound with deep figure-of-8 silkworm-gut sutures and superficial horsehair sutures. All the structures are restored to their normal position. I put in one stitch to hold the bladder up in place.

To recapitulate: When you have opened the bladder, having first anchored it with the two stitches, the next step is to estimate the condition in the bladder and the size of the prostate. Here it was exactly as we described it before the operation. There was only a small, fig-like central lobe, but large enough to produce obstruction at the trigone. The sound is in the urethra. Passing my finger along the anterior and right margin, I force it into the neck of the bladder or into the neck of the prostate; then I have the anterior urethra at that point, and get in along the anterior margin of the isthmus. After the urethra is torn I swing my finger around under the capsule and on the anterior surface of the urethral portion of the prostate. I then keep swinging to the right, keeping inside the capsule; then back to the midline. I do the same on the left side.

I cover the skin with boric powder instead of bismuth subiodid, because the former is an antiseptic under these circumstances while bismuth is not. The figure-of-8 stitches are tied there over a strip of gauze. Freyer does not leave a drainage-tube in the bladder. We do, and we use the Bremermann constant irrigation with sterile water for about seventy-two hours, depending on the nature of the case. The suprapubic drain will be removed in a week or ten days and the wound allowed to close. If there is any infection present, drainage must be maintained for a longer period of time. The urethral catheter is then also removed. It may be necessary to pass sounds later if there is any evidence of a urethral narrowing or stricture.

As to the indications for operation: The size of the prostate has nothing to do with the indications for operation; the obstruction to the outflow of urine has. The first time he has an obstruction, if it is the result of exposure, the patient is not to be operated on right away. Put him to bed, give him a hypodermic of morphin to quiet him, and the retention will disappear. The moment the man begins to lead a catheter life, no matter

how cautious you are as to asepsis, no matter how well the catheter be used, it is only a question of time when the patient will have an infection of his bladder from the use of that catheter. Antiseptic precautions count for nothing. Urethritis, from the use of the catheter, and sepsis of the bladder are bound to occur sooner or later.

In his first article Freyer said he dissected the prostate away from the urethra, and then every practitioner intended to do it himself, and found, as most of us have, that it could not be done. The fact of the matter is that Freyer showed beautiful specimens where he had removed an inch and a half or two and a half inches of the prostatic urethra.

There is no carcinoma in this case. There is merely a hyperplasia of fibrous tissue. It is difficult to get out a hard prostate. Large prostates are taken out by the suprapubic route, by the plan I have followed. We are doing four of these operations by the suprapubic route to one by the perineal. There is less injury done to the urethra by doing the suprapubic operation. If we analyze the cases that are operated by the perineal route and those that are operated by the suprapubic route, we will find that the mortality depends largely on the judgment of the operator more than on anything else. If the surgeon is careful about operating in cases of arteriosclerosis or infection, he is likely to have uniformly good results with either of these operations. If, on the other hand, he operates when there is decomposition of urine, when there is an active infection, the mortality is very likely to be high, no matter what route he selects. The mortality depends on the conditions present with the enlarged prostate more than on the method of its removal.

NEPHROPYELOPLASTY

HISTORY

Miss R., aged thirty-one, stenographer, came to hospital because of recurrent attacks of pain and swelling in the region of the left kidney.

Family history negative. Measles and whooping-cough when a child, with good recoveries. No other illness. Has a slight leukorrhea.

Present Trouble.—Fifteen years ago patient had a dull aching pain in the left side and in the back over the kidney, accompanied by nausea and vomiting. Would not get complete relief from pain on lying down. At that time she never noticed any enlargement during the attack, but there was marked tenderness in left kidney region during the attack. She does not remember any tenderness being present after the attacks at that time. Each attack would last about one hour. No chills; no fever.

Five years after the onset of this trouble she noticed a well-defined enlargement in the left side, just beneath the ninth costal arch, coming on with each attack, and remaining for from two to three weeks after each attack. The patient also noticed an increase in the amount of urine passed immediately after the subsidence of pain. For the past ten years tenderness was very marked during and subsequent to each attack. There is also constantly present a slight tenderness on deep palpation. When questioned regarding tight lacing, the patient states that she did not err. She was unable to wear corsets during each attack, but when effects passed away she is more comfortable when wearing corsets. Attacks are more frequent during last few years and last longer. Patient is now recovering from an attack which lasted two weeks. There is pus in the urine.

COMMENTS AND OPERATION

DR. MURPHY: No casts and no blood in the urine?

THE INTERN: No, doctor.

DR. MURPHY: There is a very good history. It is practically a classic history of this type of trouble. A typical Dietl's crisis, typical retention of fluid in the pelvis of the kidney, recurring at intervals, first manifested years ago and increasing in frequency and in duration. She had first pain, then nausea and vomiting, not coming on quickly, but after a considerable lapse of time. Then came the manifestation of a tumor in her side. She noticed that tumor enlarging and becoming more sensitive as it enlarged. She has diminished urination during the pain. Finally, as the tumor reaches its climax in size and the pain its climax in severity, and when the reflex symptoms are at their worst, she suddenly has a cessation of the pain, an increase in the quantity of urine, a gradual diminution in sensitiveness over the tumor, a diminution in the size and resistance of the tumor, soreness continuing for a number of days,—eight or ten,—and then a return to the normal condition. She has found that she can lessen the severity of the symptoms by going to bed, lying on her back and elevating her hips, getting her kidney down below the costal arch and getting her ureter straightened out. That is a Dietl's crisis in all its particulars.

As a result of years of distention of the pelvis of the kidney she has developed a chronic pyelitis—she has pus in the urine. She has had no elevation of temperature at any time in these attacks?

THE INTERN: No, doctor.

DR. MURPHY: This means that she had no virulent infection. What does it all mean? It means a periodic retention of urine in the pelvis of the kidney from any one of the many causes that produce it. The most common cause to produce this condition is an angulation at the attachment of the ureter to the pelvis of the kidney. The next most frequent cause to produce this condition is maldistribution of the arteries: one of the arteries, in place of passing along on the anterior side of the ureter, comes out

under the ureter and is attached to the kidney on its posterior surface. The normal anatomic attachment of the large renal veins and renal artery is anterior to the ureter always. As this artery passes around the ureter it compresses and angles the ureter, and as the kidney enlarges the artery is pushed down and becomes tighter and tighter.

Israel, of Berlin, was the first accurately to describe that condition, and his book on the Surgery of the Kidney was the masterpiece of that time. It is based on the clinical study of over 300 cases of renal surgery, and it is the best book that was ever written on renal surgery because it was based on facts, on personal observations made by one man, and I believe that all text-books of the future are going to be written on that basis. That is the proper basis on which to write books, because then you give first-hand information and give it from personal, clean-cut observations at the operating-table and at the bedside.

I cannot feel the kidney now, although I felt it some days ago.

In operations on the left kidney you want all the room you can get. You must remember, when working on the left side, that the diaphragm not infrequently comes an inch or an inch and a half below the costal margin—more frequently below in the male than in the female.

The patient is placed on her right side, with the right leg extended and the left leg flexed on the abdomen. The body is raised at the waist-line by means of sand-bags or a table which may be elevated at an angle in the middle. This brings the kidney well out from under the ribs and throws it up against the body-wall. The incision is extended downward and inward from the outer edge of the erector spinæ muscle at the level of the twelfth rib to the desired length. It must be large enough to give free access to the kidney. The abdominal wall is cut through layer by layer until the perirenal fat presents itself in the wound. This is torn through until the kidney is reached. Remember you are behind the peritoneum.

There is the twelfth intercostal nerve and the ilioinguinal. That you do not wish to cut, nor do you wish to include it in the final closing suture. If you include it in your suture, the patient

suffers intensely with pain for several days after the operation until the nerve is destroyed.

Now I commence and look for the kidney. Here it is, below where I went in—and well below. I have planned the incision so as to cause the least possible injury to the muscles. There is a mass that feels hard and firm. I wish to avoid the peritoneum, if possible. That mass should be the kidney. Its capsule resembles the peritoneum very closely. On the left side you are more likely to get into the peritoneal cavity below. The true fatty capsule of the kidney is now visible, and the kidney is shining through. I am well behind it. I must get onto the true capsule of the kidney and then displace the kidney and bring it out. This kidney should be firmly adherent to the peritoneum in front,—very firmly adherent,—because it is so movable and has been sliding around. The kidney is of very great size, with the pelvis enormously dilated. I am well under the upper pole, but I am not under the lower pole. I will just increase the size of my incision downward through the muscle margin so the kidney will vault forward from below. I am trying to get under the lower pole without rupturing the cortical portion. The delivery of this kidney is unusually difficult because of the size of the gland, but with care it is accomplished.

We have here a beautiful pathology. There is a dilatation of the pelvis that has crowded the cortex and caused a dilatation of the cortex. This is a beautiful illustration of the obstruction that occurs from malformation of the ureter the result of pressure by the vessels. The point of obstruction is close to the pelvis. It is a valve-like formation, first described by Dr. Fenger. There are some connective-tissue bands passing along below the ureter. There is no artery here doing the mischief, but the ureter is valved on the side. That is the cause of the trouble.

Will I take out that kidney now? No, there is no occasion for taking out that kidney. It is a good kidney and does lots of kidney work. I will take out all the sac with the valve, and I will reattach the ureter. I will do a pyeloplasty.

You can see now why this patient had a Dietl's crisis, and you can see what the Dietl's crisis did. I mean the condition that

produced the Dietl's crisis. First, she had a dilated sac; then, after the sac was as large as it could get, and the collateral supports from the outside made additional pressure on the sac and kept the sac from spreading sideways and downward, it had no place else to go, and it extended up into the pelvis of the kidney.

This large sac has been excised and we can now see how the valve was formed in the ureter, just as Fenger described it. We will now unite the ureter to the stump of the pelvis, but before inserting the last stitch we will pass a ureteral catheter down into the bladder to insure its patency. You will notice that, as I pass the needle through the pelvis wall near the normal renal margin, I get a little bleeding, which we wish to avoid.

Now we commence to do business. I will take this capsule and hold it in this position and show you how I will handle it. Remember I detached that ureter in the fold where I knew the valve was located. Then I cut out the portion of the ureter with the fold. This procedure was described in a paper I read before the Southern Surgical Association. I took out a V-shaped piece of ureter. No other tissue except the peritoneum lives so long and so well as does the ureter.

The next step was to turn out this kidney and take out all the sac clear down to the pelvic margin, until we reached the calices. I amputated that large sac clear down to the calices. Just when you get near the margin of the renal portion of the pelvis you have that venous bleeding and it tells you when you are there—it announces that you are at the limitation of the normal pelvis of the kidney. I closed that sac at the same time that I cut it off until I got down to the place where I reattached the pelvis to the ureter or to the pelvic portion of the ureter. I was very careful not to cut too close to the calices. This is the fourteenth case of this kind in which I have done this operation, and we have 14 patients well.

I made an entropium of the cut edge, just reversing what I do when suturing the peritoneum. If I cut too closely to the lower calyx, I would have a pocket and the operation would be a failure.

Now I let this kidney fall back into its position. We com-

mence to estimate where we are going to attach this ureter. We get this pelvic wall turned out now for the attachment of the ureter. Before I do that I must elongate and enlarge that ureter and make a different opening in it from the one that is in it now. This is the real key to the situation. The ureter must be split where the valve is. I let that portion remain on until I split it. I will sew that ureter as a piece of patchwork on to that pelvis. I am going down five-eighths of an inch below the place where it had valved. I will use a continuous suture of catgut with the knots and the thread outside of the ureter. You can see how that cannot valve or kink again, because the V-shaped patch is put in that ureter below the point where it was formerly obstructed, and it has a flat surface now. The stitches must be put in very carefully, so as to insure perfect approximation of the ureter to the pelvis. The ureteral catheter which we inserted will keep the ureter patent. I pass it out up through the cortex of the kidney and fix it there so it cannot escape. That is the model that maintains the opening in the new ureter until it becomes organically healed or repaired. We will now complete this part of the operation by putting in the posterior line of sutures. The ureter is now securely approximated to the new kidney pelvis.

When we first did this operation—it was an original procedure with us—we had some misgivings; now we have not the slightest misgivings. We know positively that the ureter is going to do its work and that this kidney is going to be saved.

I told you that aside from the peritoneum, this tissue unites more rapidly and securely than any other tissue in the body; that is why we can take such liberties with it. This was a most beautiful illustration of this condition in its superlative degree, with the kidney not materially interfered with, so far as secretion was concerned. There is a great dilatation of the lower pole of the kidney—more than of the upper. There is not so much secreting kidney tissue as normally. I am going to amputate this lower pole, because it has in it very little secreting tissue, and as a large sac it will hazard the final result, as far as this kidney contracting down to its normal size is concerned. I will run a puckering string through and close up this end of the kid-

ney. I am putting the sutures in close to the pelvis, so that any leakage that comes will be siphoned out.

Before putting the kidney back in place we will split its fibrous capsule longitudinally along the convex border, and strip it off down to its attachment at the hilum. Why? To allow the formation of adhesions which will anchor the kidney. Now I will put this kidney back into place. It falls back into position easily. I show you this nerve again—the ilio-inguinal nerve. That must not be included in the suture. First is the catgut closure of the muscularis. We are uniting these muscles accurately because, if we do not, we may have a hernia of the kidney subsequently—a lumbar hernia. I am watching that ilio-inguinal nerve with each and every stitch, so that I will not include it in the suture. Was any effort made to anchor the kidney? No, it will anchor itself. I took off the true capsule, and that will anchor the kidney by the formation of adhesions.

Let the records show that the kidney was greatly distended; that it was a little difficult to luxate it into the incision. When it was luxated, it showed a great dilatation of the pelvis and a dilatation of the calices. The greatest dilatation was at the lower pole—so much so that the lower quarter of the kidney was a sac. The dilated pelvis was first amputated, leaving a selvedge margin to which the ureter could be attached. The ureter was valved at its outlet, and it was this valving that caused the occlusion, and it was this valve-formation that gave the patient repeated Dietl's crises and caused the dilatation of the pelvis. It was this also that caused the dilatation of the lower pole of the kidney, which was exposed most to the pressure on account of the luxation of the kidney downward.

I am lifting up the muscle in the lower loop of the figure-of-8 so as to get it firmly fixed. The skin wound is closed in the usual manner. We attach a large tube to this ureteral catheter, which you remember extends from the bladder upward and out through the kidney substance. The urine secreted by the kidney will flow down the ureter into the bladder, and the catheter will siphon it back again out into a vessel which will be placed at the side of the bed.

We have also placed a rubber drainage-tube into the retro-renal fossa, surrounding it with a strip of nosophen gauze. The sponge count is correct. A very large dressing is applied and kept in place with adhesive.

The drainage-tube will be removed in a week, the ureteral catheter in ten days or two weeks, when there is reason to believe that the ureteral wound has healed. The patient will then be in good condition, so far as the operation is concerned, and can return home as soon as she feels strong enough to do so.

ANKYLOSIS OF THE LEFT ELBOW-JOINT—FRACTURE OF JOINT WITH DEFORMITY

HISTORY

Mr. M., salesman, aged forty-eight. Entered Mercy Hospital February 19, 1912, because of limitation of motion in left elbow- and shoulder-joints. Family history negative. Neisserian infection twenty-three years ago, with no apparent ill results. Was a very heavy drinker eleven years ago, and took treatment for this habit. Has used no alcoholics for the past nine years.

Present Trouble.—November 21, 1911, he was in a railway coach which rolled over an embankment. He was thrown about the interior of the coach with great force, and though he did not entirely lose consciousness, he was badly stunned. He was able to walk out of the car immediately, but found that his left arm hung limp and felt numb. He was taken to a hospital, where it was discovered that his left humerus was fractured at its upper portion, but not compounded. The bony structures of the left elbow were fractured and compounded, the olecranon being driven out through the skin.

The wound was cleansed with tincture of iodin. He states that it never discharged pus. A cast was applied, which he wore for twenty-five days. When it was removed, the shoulder-joint was nearly immovable. Motion at elbow was limited. The forearm could not be flexed or extended in an arc of more than four inches. Forearm was held almost at an acute angle. The movements of hand and wrist were not affected in any way, save for some limitation of pronation and supination. Muscles of arm and forearm are flabby and atrophied.

Examination of heart and lungs revealed nothing pathologic. Urine, normal; temperature, normal.

COMMENTS AND OPERATION

This patient had an impacted fracture of the upper end of the humerus, which has already fully repaired. That the doctor who took care of the case in the beginning did splendid work is evident from the fact that although the patient had a compound comminuted fracture of the elbow-joint,—wide open,—the doctor kept his hands and instruments out of the wound and the result is this primary union. That is something attained worth while under adverse circumstances. He has saved this man from much trouble which he would otherwise have had. At that time the arm was swollen, was severely injured, and the doctor was unable to outline the bone, and even if he had been able to outline it, I do not know that he could have done very much. The fact was that when the patient struck the point of the elbow, the olecranon was driven through the articular surface of the humerus, carrying with it the internal condyle, and dislocating it upward and inward; the outer condyle of the humerus was driven down to or through the skin. The process of repair went on, and there is now a fibrous union of the internal condyle, with the articular surface of the ulna. The external condyle is protruding away below the level of the posterior surface of the humerus. The bones of the forearm were pushed upward, forcing the external condyle almost, if not quite, through the skin. We have here a splendid result, so far as asepsis is concerned.

He also luxated the head of the humerus. There is an impacted fracture of the neck of the humerus. He has bony union, however, and fairly good position, so that it should not be disturbed. The tip of the acromion and the tip of the coronoid process bear a proper relation to each other, and he has there an articular surface without ankylosis.

It is impossible for us to elongate the internal condyle again or to bring it down to the level of the external condyle. What we must do, therefore, is to shorten the external condyle and thus bring it up to the level of the internal condyle, and make it of such a shape that the head of the radius can slide around the external condyle, preventing the head of the radius from becoming

Fig. 105.—Photograph showing deformity of elbow.

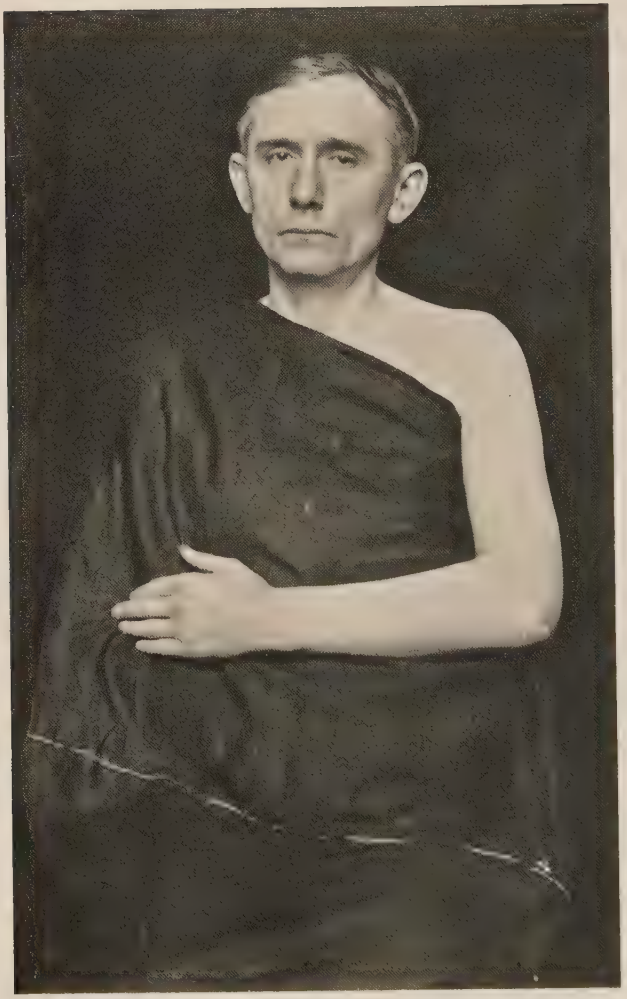

Fig. 106.—Photograph showing fixation of elbow.

Fig. 107.—Skiagram showing fracture. Marked displacement of fragments.

Fig. 108.—Skiagram showing fracture of elbow-joint through the condyles.

Fig. 109.—Skiagram showing deformity.

agglutinated to the freshened articular surface, which we must make on the anterior surface of the humerus by interposing a fascia and fat flap. In addition to that, if, when we flex the arm, the coronoid process impinges against the anterior surface of the humerus, we must correct that. If the coronoid fossa is gone below, we will either have to take off the tip of the coronoid process so as to get it to come around farther, or make a cavity in the lower end of the shaft of the humerus to permit the coronoid process to go in and not interfere with motion. We will do whatever is indicated when we get in there.

This is a complicated condition. He has been saved from an infection which would have obliterated the synovial surface in the joint and resulted in an ankylosis. Therefore we do not have to do a typical arthroplasty, only a partial one, covering the lower end of the external condyle only, in place of covering the lower end of both condyles of the humerus. The line of action is different from that which we have followed in some of the other cases we have had, because of the ankylosis we encountered there. It is usually an ankylosis the result of infection and inflammation, and not an ankylosis that is due to trauma, as in this case.

It is difficult to interpret the skiagram in this case. The anatomic surface of the articular end of the ulna has not been disturbed. That is the first thing. The internal condyle is broken off from the shaft of the humerus and displaced inward and upward. There is limitation of motion, but not a bony ankylosis. The ulnar nerve is not injured, but it is displaced by a splinter of bone which lies between it and the fractured internal condyle, showing clearly that the relation is not maintained. We must do something to permit this arm to come down. The principal reason the arm does not come down now is because there is an impingement against this external condyle, so that we shall have to take off a portion of it. That is proposition number one. Whether we shall have to produce a new cavity for the tip of the olecranon, or take off a portion of the tip of the olecranon, we will have to settle later

I am going to make my incision on the back of the arm to the

inner side of the olecranon, so as to get sufficient tissue for the interposing flap. When we rotate the radius, we find that it is situated away above, instead of almost on a level with, the condyle. The incision is about five inches long. It extends through the skin and fascia, and then the field of operation is exposed. The musculospiral nerve is in view, but we do not expect to get near it. We will, however, be obliged to stretch both the ulnar and the median nerves when we put this arm through the full latitude of motion. We have had three cases of paralysis following this stretching, none of them permanent, however. The paralysis disappeared in all of them after a time.

We will now expose the external condyle of the humerus by dissecting outward, undermining and retracting the skin-flap so as to get a good view and provide easy access. There is the articular surface for the radius on the humerus. The head of the radius is not short at all. It has gone backward. It is fractured. Next, we will dissect inward so as to expose the internal condyle and get our landmarks. I will take off a little of this external condyle, which impinges against the head of the radius, and straighten the joint line. That will make practically a new external condyle and a new articulating surface for the head of the radius—and it is here that I shall interpose a fat and fascia flap to prevent the occurrence of an ankylosis. I provided originally for a sufficiently large flap, which I will take from the outer layer of the capsule and the fatty layer of the deep fascia. I have made it of full size, so that I can insert it over the articular surface, so that there will be no tendency to bony union. These two condyles—the external and the internal—should be on the same level with each other, and the anatomic conformation of the articular surface must be preserved. Of course, the internal condyle will not be disturbed in any way. Therefore, we are really shortening the arm, and while he will not have a good-looking elbow, he will have a good joint, with full range of motion.

The next step will be to cover all that raw bony surface, so that none of it will become adherent to the radius or fixed in any way. I am going now to transpose the fascia so as not to interfere with the contractile power of his triceps. I will cover these

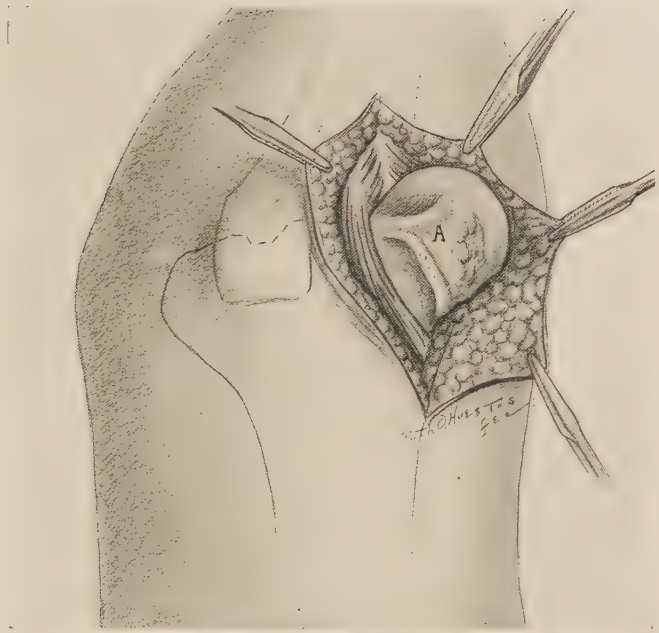

Fig. 110.—Hypertrophied external condyle of humerus (A) exposed.

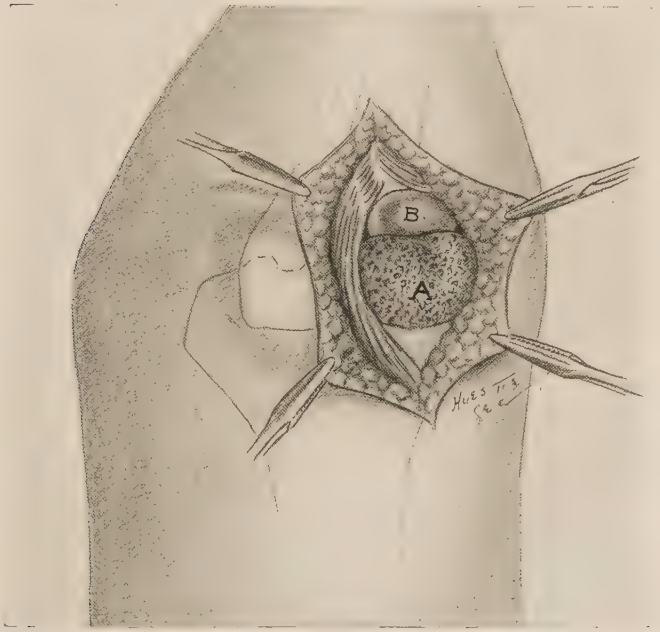

Fig. 111.—A, Redundant condyle chiseled off; B, head of radius.

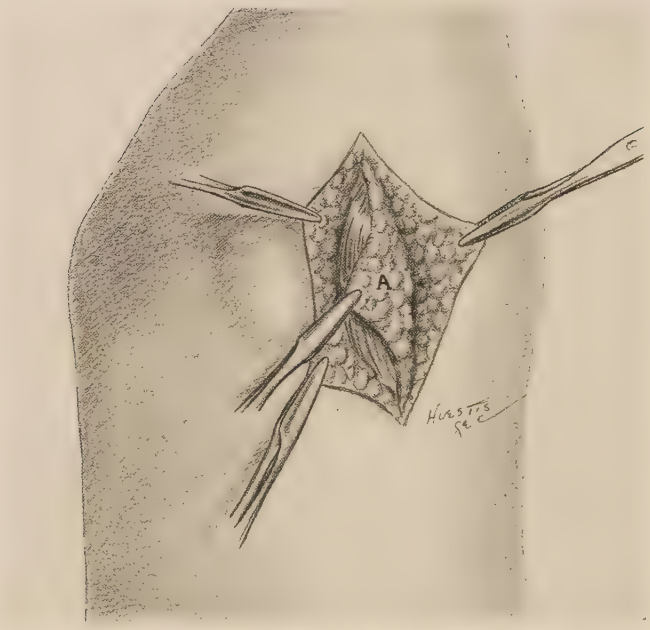

Fig. 112.—Flap of fascia and fat (A) drawn over condyle.

Fig. 113.—Fascial flap sutured in place.

exposed surfaces in such a way as to bury the head of the radius. We do not have to take off the tip of the coronoid process nor the tip of the olecranon. They both came out in good position, without the hazard of impinging, after our remodeling work on the humerus, so that we are safe in letting both remain as they are.

In order to get full extension following such a trauma as occurred in this case one must either shorten the upper end of the radius or remove the obstruction that is produced by the condyle. When the head of the radius has been luxated and has become locked, one of the classic procedures has been to remove the head of the radius, shortening the bone down to the neck. That reduces the strength of the arm very materially, although technically it is not a difficult operation to perform. A procedure that does less injury is reduction of the external condyle. The anterior half of the external condyle can readily be removed and not interfere with the function of the joint in the least, or reduce the strength of the forearm. That is done in exactly the same way as we did here, except that only the anterior half is removed, so as to permit the head of the radius to slide along the articular surface of the external condyle, and in that way getting a full extension. You can see that we have here, without forced extension, a full and free range of motion. The flap is sutured in place with phosphor-bronze wire, such as we always use for this work. There must not be any pressure on the flap or it will die, and the object for which it is put here—to prevent ankylosis—will be defeated. The skin incision will be closed with horsehair and dusted with bismuth subiodid powder. There is no danger here of paralysis of the nerves, and there is no reason why we should not obtain a good final result in this case. The cases in which we have had paralysis, as I have mentioned before today, were annoying in the length of time it took to get restoration of function—sometimes many months. See the ease with which the arm comes down and goes up! There is full motion without any effort. It will be dressed in the flexed position and placed in a plaster-of-Paris cast. This cast is made in the usual manner, being split after the plaster has set, so that it will not

constrict the arm when the tissues swell. The internal condyle was not disturbed at all. Elongation of the arm by bringing the internal condyle down on a level with the external condyle was an impossibility at this time.

We use antiseptic gauze dressings in these cases. The first dressing consists of 5 per cent. phenol gauze. The antiseptic dressing is put on for the protection of the secretion that escapes from most of the bone operation wounds. Unless you protect these wound secretions which go into the dressing, the air comes in above and below where the dressing is soaked and decomposition occurs. It is a saprophytic decomposition. This saprophytic decomposition sets up a pyocyaneus infection or a saprophytic infection, and that may extend along that wide zone that has bloody secretion in it and cause infection of the wound. We resort to the antiseptic dressing for the protection of the secretion that escapes between the stitches. You see the reason for using carbolized gauze: it prevents the secretion from acting as a culture-medium.

If that flap should necrotize, we will do a plastic operation, but we are not in the least afraid of that. We have had only one case of bony fixation following operation, and that was of the elbow. It was a case of bony ankylosis. The usual arthroplasty was done and successfully. However, the patient, a very neurotic female, would not permit of any manipulation of her arm afterward. The result was that the elbow again became ankylosed, but not because the operation was a failure.

In this case the cast will be removed in ten days or two weeks, the stitches taken out if the wound has healed, and passive motion begun. After each manipulation the cast will be replaced for a while until it can safely be left off, after four or five weeks. The patient is encouraged to secure elbow motion, so that he will have a useful elbow. [When seen last, three months after operation, patient had a very good range of motion. He could flex the arm to better than a right angle, and extension was almost complete.]

TUMOR OF THE ABDOMEN—RETRO-PERITONEAL SARCOMA

HISTORY

This patient is a physician, fifty-three years of age. He entered Mercy Hospital March 11, 1912, because of severe pain in the lower lumbar region, a high fever, and great prostration. There has been no malignancy, syphilis, or tuberculosis among his relatives. His health has been excellent up to the onset of the present trouble. At the age of nineteen he had scarlet fever, followed by albuminuria for eight weeks. He was attended by Dr. Osler, who assured him that his recovery was perfect. At the age of twenty-three he had typhoid fever severely. Eighteen years ago he suffered much from very severe headaches and nausea. He, a physician, states that this was migraine, and that his mother had similar attacks. He denies syphilis.

In 1894, when thirty-five years of age, he began to have repeated attacks of intense colicky pains in the region of the gall-bladder. Vomiting always followed rapidly, and the next day a deep jaundice would appear, lasting for from three to five days. The pain would come on very violently and very suddenly. He never had chills or fever. For thirteen years he had these attacks and became a victim of the morphin habit, often taking as high as 12 grains daily. He dreaded operative treatment, always delaying it hoping that each attack would be his last.

April 9, 1910, he was operated on by Dr. Murphy. The findings were as follows: Gall-bladder, duodenum, stomach, omentum, and hepatic flexure of colon all were adherent. No pancreatic abscess. There was one large stone in the cystic duct, and the common duct was full of stones which could be moved back and forth into the cystic duct. Stones were removed from

the ducts, each of which was the size of one's finger. A T-shaped tube was inserted, passing into both ducts.

No evidence of acute sepsis was present. Three rubber drains were used in gall-bladder, common duct, and peritoneal cavity respectively.

After the operation he recovered without incident and left the hospital May 15, 1910. For one year he enjoyed perfect health, gaining 70 pounds in weight and ceasing the use of morphin.

In March, 1911, a hemorrhage occurred into the retina of his left eye. A leading physician of St. Paul, Minn., diagnosed kidney disease, having found a blood-pressure of 260, but nothing abnormal in the urine. He left Minnesota, going to the milder climate of Florida within two months following his retinal hemorrhage. He adopted a non-nitrogenous diet, and for ten months felt very well, though the blood-pressure remained between 180 and 200. In the latter part of January, 1912, he became suddenly very ill, with a high fever, delirium, and a severe diarrhea. The fever often rose to 104° or 105° F. The urine never contained blood-casts nor was it increased in total amount. The stools were persistently diarrheic, but never were copious, offensive, or pale, and never contained undigested meat-fibers, fat, or visible blood. At first a diagnosis of dengue was made by able men. The prostration was great from the first. He had no pain until two weeks ago. He states that at this time an intense boring pain appeared suddenly in the back, just above the posterior iliac spines. It seemed to be almost in the middle. It was entirely unlike any pain he had had associated with his gall-bladder attacks, and more severe than any pain he had known. It has persisted with remissions up to the present time. He has used morphin freely since its appearance. He has not been jaundiced, nor has he had emesis during this sickness. He has lost fully 50 pounds in weight during the last six weeks. On palpation of the abdomen a mass could be felt in the position of the tail of the pancreas. It pulsated, but this was not of the expansile type. Temperature on entering the hospital was 101° F. On the morning of operation, 99° F.

Laboratory Findings, March 12, 1912.—The urine contained a moderate amount of albumin by ferrocyanid test only. It contained no sugar. One granular cast was found. Six red blood-cells were found in two slides of the centrifugalized urine. Only an occasional pus-cell was present. Chemical tests for blood in stools failed. The Widal test was negative. Blood count: Leukocytes, 10,600; differential count, polymorphonuclears, 74 per cent.; small mononuclears, 23 per cent.; large mononuclears, 2 per cent.; no eosinophiles were found in a count of 200 cells. Erythrocytes, 4,116,000; hemoglobin, 70 per cent. Blood-pressure, 184 mm. of mercury.

COMMENTS AND OPERATION

As you see, there is a very peculiar history. He went along all right after the operation for gall-stones, when he saw a doctor who made some kind of an unfavorable prognosis because of pain in his abdomen. I do not know what it was. Immediately after he had this unfavorable prognosis made he went back to the morphin habit, which he had contracted previously, taking hypodermic injections, and he took considerable quantities of the drug. Then he lived in the South, a different type of life, doing nothing, resting—when suddenly he had an elevation of temperature. How much pain he has I do not know. He cries out all the time, but whether this is morphin craving or whether it is real pain it is impossible to say. We did not, however, cut down his morphin enough for him to have pain.

This mass to the left of the midline is extremely sensitive. It does not appear to have expansile pulsation. His urine findings are not in favor of an infection in his kidney which might cause this condition. This mass is thought to be his left kidney displaced downward, which puts it in the position of the tail of the pancreas. The patient up to the onset of his illness was a very hard worker and had an influential position in his community up to the time of contracting this drug habit. He did a thing that a doctor should never do—take his first hypodermic of morphin if he has a pain, especially if he has a chronic trouble. Often doctors give hypodermic injections to women during menstrua-

tion in fact, doctors give them so often that the thought makes me creep. The frequency with which anodynes and stimulants are given to relieve pain during menstruation is appalling, and the number of victims it leaves is enormous. A man should be held responsible in some way, it seems to me, for starting patients on this downward course; to many it is worse than death in the end, and I feel we cannot be too emphatic in condemning the practice of doctors taking hypodermic injections of morphin themselves or giving them to their patients for chronic troubles.

We will open the abdomen in the midline in the usual manner, making the incision extend between the xiphoid cartilage and the umbilicus. There is the umbilicus; when this is lifted there is the mass. It is hard and pulsating. There is nothing to indicate that it is an aneurysm of the renal artery. Why did he have this high temperature? He had a diarrhea, and *following* the diarrhea he had his pain—not so much during the diarrhea. I am going in behind that mass. It is movable and resting on the vessels. It pulsates, but there is no bruit palpable. I want to examine this condition above to see if his temperature elevation has any definite relation to his old condition. There is no evidence of tuberculosis in his family. The mass is not the kidney. It is in the region of the tail of the pancreas. It is behind the mesentery, and low down. It is not in the pancreas. I am displacing the small intestine so as to get a better exposure of this mass. There it is. It is a sarcoma—and of the pulsating variety. There is the mesentery of the intestine on the side. To the right is the abdominal aorta.

Visiting Surgeon: What is that mass on the other side, doctor?

Dr. Murphy: There is no mass on that side. That is higher up, and did not originate in the pancreas: it originated behind the peritoneum—retroperitoneal, because it is on the under side of the great mesentery, and it has elevated the posterior peritoneum on the under surface of the intestine and the latter is adherent to it. The abdominal aorta is to the right of the neoplasm, and the neoplasm pulsates like an aneurysm. It is not expansile. There may be elevation of temperature with

sarcoma without infection, even a high temperature. I called attention to that many years ago.

The first case of this kind I saw was in Dr. Fenger's service during my internship in the Cook County Hospital. The patient had a trauma of the knee. He worked that day and the next day, and on the third day he had a high temperature. When he came to the hospital that night he had a temperature of 104.5° F. It remained as high as that for many days. It looked to us as though he had an osteomyelitic infection in the bone near the knee. Dr. Fenger was not satisfied about it. In a few days the mass began to pulsate; then a diagnosis was made by Dr. Fenger of an acute aneurysmal type of sarcoma. In a few days more a distinct bruit could be heard over the internal condyle of the femur, and in a few days more his leg had to be amputated at the hip. There was a distinct aneurysmal formation.

This case was the first to call my attention to the high temperature which may be associated with acute sarcoma. Our patient here began in this way. His first symptom was the high temperature, no particular pain; then pain in his side manifested itself and it became rapidly worse.

VISITING SURGEON: How would you explain his retinal hemorrhage and high blood-pressure, doctor?

DR. MURPHY: I presume it was the usual high blood-pressure accompanying a chronic infection. For years he had an infection in his gall-bladder. Most high blood-pressures are sequences of some previous infectious condition, usually of a chronic type. His gall-bladder was infected for years before he was operated on the first time. His morphin habit interfered with his assimilation, and the retinal hemorrhage is believed to be due to that. It is not at all settled as to why people get high blood-pressure and get it early, any more than it is at all settled why some races are more prone to arteriosclerosis than others. The Hebrew race is predisposed to arteriosclerosis, and it occurs very early in life. I have seen it as early as thirty-two. This is not at all the condition with the Germans—not at all the condition with the Americans, though they get it much earlier than the Germans do. The claudication manifestations, for

instance, that come with the ischemias of the leg, and especially flat-foot, are practically a Semitic disease. We see it very frequently in these people.

I remember one man who had an arteriosclerosis involving his right lower extremity first, and then the left. Finally double amputation had to be done. I made one of them, and the other was made by another surgeon. He also was treated for four years for left renal colic by very able men. He had a recurrence of the renal colic, and then I saw him in consultation with Dr. Sippy. Dr. Sippy asked me to operate on him for an obstruction in his left ureter, and I concurred in the diagnosis. He had passed no urine for six days preceding the operation. He had great pain in his left side, colicky in character, resembling renal colic pains, such as he had had many times before. We looked him over and we concluded that he had a renal calculus with sympathetic anuria in the right side, as described by Dr. Bevan. I cut down on his left kidney and exposed it; lifted it out and peeled off its capsule. It did not bleed. I cut into it and it did not bleed. It was necrotic and dead. He had an arteriosclerosis of his renal artery, with spasmodic closure which gave him the symptom of renal colic, and then his kidney, for want of nutrition, became gangrenous. He died nine days from the onset of his disease from uremia. Autopsy showed that he had a complete occlusion of both renal arteries, the result of an arteriosclerosis with secondary thrombosis. The occlusion was produced gradually, caused by an endarteritis obliterans, which is the type of disease these people have. It was the cause of his death.

I believe the flat-foot which these people have frequently is the result of an ischemia—probably in a large percentage of the cases. You will find that many of these people suffer from flat-foot, claudication, and inability to walk any distance without pain.

This man, instead of having the gastric crises that are often a manifestation of arteriosclerosis, had a renal crisis which comes with endarteritis obliterans.

It is very interesting to follow that class of cases. Here is a comparatively young man, fifty-three years of age, with an

arteriosclerosis, although this condition is not uncommon in the young. I remember the case of a prominent merchant of this city which was another one of that type—apparently perfectly well, always attending to business—a high-tension man. He had to be operated on for acute appendicitis. He stood the operation without a particle of trouble. In three days he was on a back-rest. He said to the nurse, "Will you kindly move those flowers from that position to that side. I think they will look more beautiful. Madam will be here in a few moments." The nurse lifted the flowers, took them from one window to another, a distance of not more than five feet, and when she turned around he was dead. Autopsy showed an endarteritis obliterans involving his coronary artery. He had a myocarditis of ancient origin and white streaks all through his myocardium.

Another man about fifty-one years old, head of the greatest bond house in the West,—a high-tension man,—had endarteritis obliterans or arteriosclerosis of his coronary artery. He had repeated short attacks of angina, always occurring just at bedtime. In some of these attacks the pain became more severe. In one of these he collapsed, became cyanotic, and died some fifty hours afterward. I examined his heart in this period and the expression which I used at that time was that the heart seemed to be in China, it was so very weak. During the spasm he had a thrombosis of his left coronary artery which caused his death. That accounted for the feebleness of the heart-beat during the time intervening between the onset of the thrombosis and his death. The only lesion was in the coronary artery—just one single artery. He had exactly the same manifestations that all these cases get. For six months he had pain at night before going to bed, and always at the same hour—so characteristic of these lesions; always pain in his chest and difficulty in breathing. He came in from the theater one evening, and while taking off his clothes he had a sudden severe cramp and fell to the floor. Usually the patients die instantaneously.

We shall not attempt to remove the tumor in this case, because of the absolute futility of such a procedure. The patient's life would not be prolonged. The peritoneum will be closed with

a continuous catgut stitch, everting the cut edges so that the inner smooth surface will be in contact. Figure-of-8 catgut sutures approximate the deep and superficial fascia, and horsehair closes the skin wound. No drain. Bismuth subiodid powder and a collodion dressing protect and seal the wound. The stitches will be removed about the tenth or twelfth day. [About eight or ten days after the operation this man complained of a sudden severe pain in his abdomen. He died within three minutes—before any one could get to him. I believe there was a rupture of the sarcoma. An autopsy was not permitted. —J. B. M.]

CONCUSSION OF THE SPINE WITH IMPACTED FRACTURE OF THE VERTEBRÆ

(Case Shown March 14, 1912.)

On January 17th 200 pounds of meat fell on this man's shoulders while he was in the stooping position. The shock rendered him unconscious for half an hour. When he regained consciousness he found that he could not walk, but he says that when he touched his legs he could feel them. That means that sensation was not paralyzed. He had paralysis of motion for thirty-six hours, and then he began to move his legs, and gradually has moved them more and more since. This is a beautiful case of concussion of the spine with impacted fracture of the twelfth dorsal and the first and second lumbar vertebræ, causing this apparent kyphosis, and resulting in concussion of the cord. Now, because he has only paralysis of motion and not a paralysis of sensation, we know at once that his legs will regain their motive power. The cord has suffered only a contusion. The trouble is in the zone of the conus, and not in the true cord. The axons are covered with neurilemma outside of the cord, and are fully capable of regeneration after contusion, concussion, or division if they are contacted, while if the lesion involves that part of the cord where the axons have no neurilemma, then there is no capability of restoration. Once the true cord is cut, it never regenerates, no matter how accurately it is approximated. This man is having a softening or osteitis traumatica of his vertebræ, and unless he is given mechanical support to his back he is going to bend over forward. While the process of repair is going on, until there is complete ossification of the bone, he should have a mechanical support to hold him straight, and that mechanical support should be a plaster cast.

This man did not get a metastatic infection with tuberculosis

with his trauma, as he would have if he were a child. A child that receives that kind of an injury always has tuberculous osteitis; children not infrequently get tuberculous osteitis resulting from trauma, which begins in the anterior or posterior portion of the body of the vertebræ, depending on the position in which they are struck. This man was struck on his shoulders; the anterior parts of the bodies of his vertebræ were injured. If he had fallen on his face and had been doubled backward, he would have a lordosis. I remember a patient who came to me for diagnosis. While walking one night in a stable he caught his foot in one of the lines across the pathway. He was thrown forward and struck on his hands. He got up and did his work. He got a pain in his back, but paid no attention to it. In five or six weeks it began to get worse. Liniment was put on for what was called "lumbago." He went on and was treated and treated, and finally, after a year and a half, he came here. He had a typical lordosis. An abscess developed on the side of his spine, deep in. We aspirated and gave him tuberculin. He went through the regular treatment for tuberculosis of the spine; his case was a falling forward and doubling of his spine backward. The injury in this case was posterior.

This man will have to wear a plaster-of-Paris cast for a long time. If it is taken off early, his condition will get worse. His paralysis was mild. Occasionally you have another type develop in this same relation—where the individual strikes on the buttocks, side, or on the shoulders. The effect is the same. You cannot have an immediate severe trauma of the spine without paralysis, and paralysis can commence to develop in ten days or two weeks, or three or four weeks—that is, complete paralysis.

I recall a case I saw three weeks after the injury. In five weeks he had complete paraplegia—bowels, rectum, and everything. We made a favorable prognosis, and in fourteen months he was walking; in two years he was back at his work and has been there ever since. That occurred in 1886. It was one of my first cases of the kind and called my attention to the significance of that type of injury.

TRAUMATIC EPILEPSY; DECOMPRESSION

HISTORY

This patient is a coachman, forty-six years old. He entered Mercy Hospital February 29, 1912, because of partial paralysis of right side of body, speech difficulty, and periodic attacks of unconsciousness, resulting from a blow on the left side of the head. He denies the existence of syphilis, alcoholism, epilepsy, or other nervous diseases among his relatives. He denies all venereal diseases in self. His past life has been normal in all respects and free from disease or operations.

Present Trouble.—In September, 1910, the patient was struck on the left side of the head, four inches above the ear, by a five-pound hammer. The blow was glancing, and he was not rendered unconscious. He was able to walk away from the scene of injury. About one hour afterward he found that he could not move the muscles of the right side of the body as usual. He thinks the power of movement in right arm was entirely gone, and that he could move the right leg but little. The doctor told him that the skull was fractured and scalp wounded slightly. Four hours after the injury he was operated on, but does not know what was done save that the skull was opened. Five weeks later he could move the hand a little and movement in face was much improved. All movement became better very slowly, and he could do his work and walk about, though his leg dragged somewhat and he could use his arm to only a limited extent. He could bring it up to a level with his shoulder. The leg seemed less affected, according to his statement. Following the injury his speech entirely disappeared, but after the operation he could talk a little, but with great slowness, especially at first.

November 15, 1911, he suddenly lost consciousness and thinks that others said he was asleep, and could not be wakened for

fully half an hour. Afterward he felt dizzy and had a queer feeling in his head for twenty-four hours. He knows nothing at all in regard to convulsions or muscular spasms at this or at any other time.

February 26, 1912, he knows that he again "went to sleep" and could not be awakened. He remembers very little about it except that he had a queer, dizzy feeling in the head. He never vomited or had severe headache at this or at any other time. Temperature is normal; urine, negative.

COMMENTS AND OPERATION

This is a fairly typical history in every way. It is typical in the fact that the man had an injury; that he has had a depressing injury, which is not always the case; that he had a paralysis of speech and paralysis of his right hand and in part of his right leg. This has materially improved. His speech has materially improved, but his right hand is still somewhat incapacitated. It is the center of disturbance.

The immediate effect of injuries of this class is recovered from, as a rule. Handled in anything like a careful aseptic manner, even with drainage, they recover, and they recover without drainage. But the immediate recovery is only a part of that which is desired in these cases. Next comes the restoration of function in the impaired convolutions of the brain, and we know that we have many associated centers; that a center may cease to functionate for a short time, or even be removed, as I have seen a spoonful of brain tissue removed, and the function of the convolution, as we understand it from a physiologic standpoint, be eventually restored either completely or partially.

But that is not all. The important thing in every head injury, whether it is a compound fracture or a simple fracture, or whether it is a concussion of sufficient severity to produce unconsciousness, all centers around the fact whether the patient is subsequently going to have epilepsy, or whether he is subsequently going to have these epileptic convulsions and finally become a victim for the asylum. That is the important point.

The injury received does not need to be a hammer-blow: it

can be any blow on the head that produces unconsciousness. For a long time we believed that the Jacksonian type of epilepsy was the sequence of a compound fracture, and rarely ever the sequence of a concussion. We are now learning that a concussion very frequently results many years later in epilepsy, and that led the late Dr. Fenger, who was the father of it, to adopt a treatment which he felt would obviate the ultimate epileptic convulsion.

There is another lesion which is a sequence of trauma not associated with fracture, frequently not even associated with unconsciousness, and that is an epidural or subdural hemorrhage, with the formation of an encapsulated cystic condition, either outside or inside the dura. This gradually produces an increasing effusion and increasing irritation, and finally the patient commences to show, if not convulsions, at least changes in his mental condition, and he finally lands in an asylum, never having had a depressed fracture or anything more than a contusion or a concussion. In some of these cases you have momentary stunning of the patient. The patient's scalp is most always not cut. He does not have the period of reaction immediately after the injury and subsequent unconsciousness that one has in the middle meningeal hemorrhages. But he does have a circumscribed cystic formation, hemorrhagic, we believe, frequently later of a serous or grumous character, with a mass of connective tissue around it, slowly increasing in size, and, as I have said, the patient winds up in an asylum. Preceding the disturbance of his mind he has repeated attacks of headache, these attacks increasing in severity. That, too, is very common with a traumatic epileptic patient. The period of time that may elapse between the injury, from our personal observation, where the injury is clearly the etiologic factor, ranges from a few weeks to over thirty years. In one patient it ranged over thirty years, in another, twenty-seven, in another, twenty-six, and in many, twenty-five and twenty-four years, but the greater number appear under twenty years after the accident. That has been one of the important factors in giving a certificate to an individual who has received an injury to the head sufficient to make

him unconscious. To show you how much stress I place on that I have often said that rather than be rendered unconscious by a blow on the head I would prefer to die—so much fear have I of the secondary lesions that occur in connection with these injuries.

This man had a depressed fracture, as you see, as it stands out when he is under the anesthetic. Before he was put under the anesthetic there was a pronounced stellate depression, the same as you see it here.

In the management of a case like this we usually make a U-shaped flap wide of the lesion, and expose the margin of the bone. I am not going to take out a bone-flap, but make a subperiosteal removal of the bone. You see the margin of the bone. I will enlarge the margin as much as I think it should be enlarged, but I will probably remove a considerable portion of the dura.

The question comes up in all these cases as to why we have the epileptic convulsions. Are they due to the irritation of the dura, or are they due to irritation of the brain. That has not been definitely settled, but it is my conviction, from clinical observation and from some experiments made many years ago, that it is the irritation of the dura that causes the convulsions. I remember a case that occurred when I was a student in Rush Medical College, at which time a celebrated French physiologist made a demonstration of convulsions occurring in a dove from injuries to the dura. Now, the dura will be adherent to this. I want to preserve my skin-flap. I want to keep within the proper zone. I am deep down on the dural tissue, because subsequently I shall remove a portion of tissue that rests immediately beneath this scar, because it is my belief that the convulsion is due rather to the cicatricial tissue and irritation of the dura than to the irritation directly of the brain tissue itself. There is the cerebrospinal fluid, as you see, and here is the dura. I shall lift that up. The dura is involved in this large mass of cicatricial tissue. There, I believe, is the cause of the convulsions. It is the irritation here which I believe produces the convulsions. Now I am going to get under the adherent margin of bone. This attachment shows how little of the osteogenetic force we have in the internal layer of the periosteum, that is, in the predural

periosteum. It shows that we have but a few of these feeble osteogenetic cells, differing from the osteogenetic force of the periosteum in other positions of the body. You will see, it has not attempted to span this gap; that is, it does not build bone, whereas in the tibia and femur many times you will find it spans a gap an inch and a half and even two inches, frequently an inch, and very commonly smaller distances. I am turning down the external periosteum just above the beveled zone, so as to get under the predural periosteum without any danger or difficulty. As soon as I have this flap raised from the margin I will have no difficulty in separating it for a greater distance. The only bleeding we will have will be from the veins of Santorini, which one encounters in doing the Gasserian operation.

I have detached the dura from its fixed position all the way around. There has been no tendency to hardening here. There has been a depression that we believe is due to the contraction of the tissue. Now comes the important factor. In our management of these cases we have found that with the removal of this irritating zone of the dura we obtain the best results that can be obtained by any line of treatment, not merely detaching the dura from the bone, but excising it, and then we will interpose a thin plate of paraffin, preventing adhesions from forming. This coal-tar paraffin is non-absorbable and non-irritating, and remains permanently of the same size and shape, differing from the whale-oil paraffin, which is a normal product and absorbable. The coal-oil paraffin becomes encapsulated with a thin layer of connective tissue. I go clear out to the margin of cicatricial tissue, and free it for a little distance back on either side. You see how dense and firm that is from the pressure which is required to cut it with this high leverage scissors. There is the central cicatricial mass, and here is the connective tissue beneath it. There is the cortical substance. We have taken out all the cicatricial portion of dura, and in removing that we have removed the great source of danger. This man is at an advantage in being operated on at this time, namely, he has only had two convulsions. He has not had the epileptic habit established. In the second place, he has the advantage in that the

force of the blow was concentrated over a small area, differing entirely from a man who has a fracture of the skull with the force of the blow distributed over a very large area. I force this paraffin in underneath the skull on either side. That protects the brain from attachment to the dura or scalp, all over the injured zone.

In some operations that have been suggested in cases of this kind a considerable portion of the cortex has been removed. The cortex comes up clear, not cicatricial, perfectly soft, and the removal of that would be detrimental and would add nothing to the percentage of cures of the epileptic attack. We have had lots of experience with paraffin, but not so much in putting it in as in taking it out, particularly when operating for hernias that have had paraffin injected, and we have had to remove the paraffin subsequently because it did not cure the hernia, and we know what a thin layer of connective tissue was deposited around it.

In estimating the treatment for ankylosis we have figured a number of times that if we could get a material like paraffin that would not be absorbed, and, at the same time, that would not become vascularized or infiltrated with connective tissue, and would not fracture from pressure, we would have an ideal interposing material in many of the joints, if it would stand pressure. The trouble with paraffin is it does not stand pressure. If you put pressure on it and it is cold, it will break. If you put pressure on it when it is warm, it will press out. It would be desirable if we could find material that would not do this. To remold it down into conformation is not a difficult proposition. The interposition of the flap and retention of the flap, with its vitality, is a difficult proposition, and adds most to the obstacles we have in getting good results in these cases of ankylosis, because of the quantity of connective tissue that forms around the new flap and the joint where it was placed.

We never drain these wounds. When we operate for a trifacial lesion, where we have such a large quantity of blood escaping into the cranium, when one is closing it, in fact, when there is a blood-clot an inch in thickness almost, one has no

fear at all, but let it form, it is rapidly absorbed, and it does no harm.

Let the record show there was a cicatricial mass one-eighth inch in thickness in some of its area, covering over the exposed portion of the dura; that it involved the pia, and in some places the convolutions; that this was freed first from the scalp and from its bony margin over one-half inch all the way round. It was then excised on a line with the bony margin, taking off just the surface of the convolution. In the second place, sutures were used to stop the bleeding, which was very slight during any part of the operation. A plate of paraffin one-tenth inch in thickness was then inserted on the surface of the brain and under the bony margin. It was adjusted to give it pliability. It was gradually worked under the margin of the bone, so that it separated the dural margin from the bone and was elevated in the center to the level of the normal bony elevation, and that it was a little lower under the margin. When this was inserted, there was no bleeding. Let the records show that the dura could not become adherent to the lacerated or fractured edge of the bone, and that there was no opportunity for the dura to become subsequently attached to the scalp.

May 10th.—This man's speech is improved 70 per cent. over what it was. He can use his arm much better, and has resumed his occupation as a coachman. He has had no headaches or convulsions. His employer, a physician, believes that with his present rate of progress the patient will be completely restored in three months.

TRANSPLANTATION OF BONE

Osteitis Fibrosa Cystica

HISTORY

(Written June, 1911)

B. K., male, aged eleven. Family history negative. Had measles; otherwise always well.

Present Illness.—In September, 1908, he was run over by a bob-sled, the hind runner running over his right tibia at the middle of shaft. Had severe pain and swelling over that area. There were no fractures. Patient remained in bed for one week, when the pain disappeared, but some swelling remained. One year ago his mother noticed that the swelling was gradually increasing in size, but the patient did not complain of any symptoms; no pain, no chills, and no fever, and he did not favor the injured leg.

Four weeks ago patient slipped on a stone curbing and struck the same area of right tibia on the curbing. He had severe pain in bone and swelling rapidly increased in size. A week later he was operated on. The bone was cureted and a part removed. Mother took a specimen of bone to a Chicago hospital, where she was told it was a spindle-cell sarcoma. After the operation, three weeks ago, patient made a rapid recovery, but swelling still remains. There is also slight pain. No chills or fever. No loss of weight. No night-sweats and appetite good.

First Portion of Operation by Dr. Golden.—An incision was made over the left tibia, a portion of the tibia 9½ inches long by 1 inch by ¾ inch was removed from the crest. The periosteum was removed from this bone. The bone was laid aside, being very careful about handling it. The periosteum was sutured with catgut over the 9½-inch trough, where the bone was removed and the skin closed with horsehair. A collodion dressing was applied.

Second Portion of Operation.—An incision was then made over the right tibia, near the tumor mass. Eight and one-half inches of the tibia was removed with the tumor. Each end of the bone, upper and lower, was notched out, and grooves were made in which to place the ends of the bone to be transplanted. The sharp and roughened edges of the distal and proximal ends of right tibia were removed with bone-cutting forceps. The portion of bone 9½ inches to be transplanted was then placed in the gap in the right tibia, its ends being placed in the holes made in each free end of the right tibia fragments. Measurements were carefully taken, so as not to get any shortening, and two nails were then put through the proximal and distal ends of the transplant and tibia to keep it from slipping into the tibia too far. Muscles and fascia were then sutured over the bone with fine wire. Skin closed; collodion dressings put on. Each leg was put into a plaster cast extending from the toes to about the knee-joints.

OPERATION BY DR. JOHN F. GOLDEN; COMMENTS BY DR. JOHN B. MURPHY

Dr. Murphy: How long after the trauma before the swelling was noticed?

The Intern: One year.

Dr. Murphy: How do you figure that? The accident occurred in 1908, and you say one year ago, and it is four years since 1908. How do you figure that? What has become of the other years?

The Intern: This history was written two years ago, Dr. Murphy.

Dr. Murphy: All right; that clears it up. Go on with the history, doctor.

Dr. Murphy: When was the first operation done?

The Intern: In July, 1911, by Dr. Golden.

Dr. Murphy: Yes, that is right, Dr. Golden did the operation at that time. He took out all the bone in which the cyst was situated. How long was the transplant?

The Intern: Eight inches.

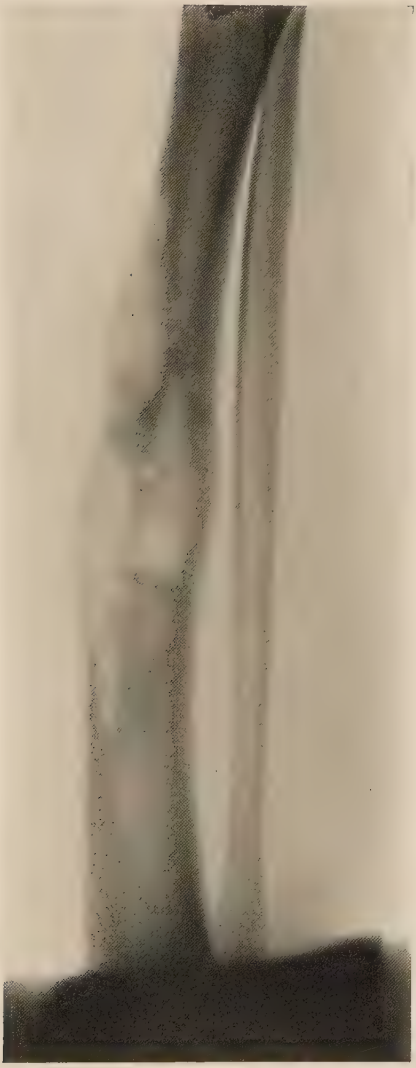

Fig. 114.—Skiagram showing tumor of tibia before first operation.

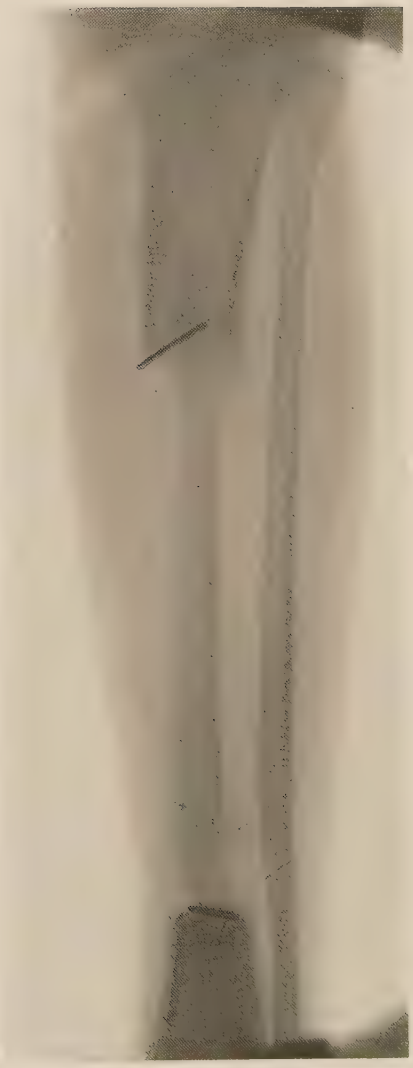

Fig. 115. -Skiagram showing transplant after first operation.

DR. MURPHY: Eight inches? Is that right? It seemed to me it was 9 inches or 8½—which was it, Dr. Golden?

DR. GOLDEN: Nine inches and a half.

DR. MURPHY: That is an illustration of one of the cases in which we have done this bone transplant work. Dr. Golden did this work during my illness a year ago and he succeeded in getting a full growth of bone, except at the lower end, where the nail was put in. The skiagram shows that there was a little suppuration at that point, and that there was a little necrosis and absorption. It was not of the extreme suppurating type. This bone has regenerated beautifully.

There is the splendid result Dr. Golden got with a transplant 9½ inches long placed into the space from which he removed the diseased portion, which was diagnosed first by one pathologist as a spindle-cell sarcoma and by a second pathologist, subsequently, when the whole mass was taken out, as osteitis fibrosa cystica. That just shows us how significant the microscopic diagnosis may be, or, rather, may not be. We have come into the habit of accepting that as final, but it is not final at all.

From the gross appearance of the skiagram before the operation one would conclude that it looks more like an osteitis fibrosa cystica than like a sarcoma, and that picture is more valuable than any picture that the microscope shows, because sarcoma of the spindle-cell variety, of the large round-cell variety, and of the small round-cell variety destroys bone in a definite way. The chondrosarcomata destroy bone in a definite way, so that you can draw your conclusions just as well from the skiagram as from the microscopic picture, and you can form a fairly accurate estimate before you start your operation and before you remove a portion of that bone as to the line of procedure you are to follow.

If this diagnosis of round-cell sarcoma is correct, then amputation is the only procedure that should be done at this time, so far as we know; if, on the one hand, it is a large spindle-cell sarcoma or a giant-cell sarcoma, excision will probably give a final cure. If it is a chondrosarcoma, there is about an even chance of removing the bone and getting a final cure. A chondrosarcoma will recur as a metastatic sarcoma in some

other position in the body. Therefore, the conservation of the limb, instead of amputation, is the procedure that should be attempted in all these cases, and that has led to the large number of cases we have had of removal of great areas of bone, and to a complete and perfect reproduction of bone following transplantation.

In Dr. Golden's case there is complete healing in of the lower end of that bone, but a non-union. This boy must have a solid bone from one end to the other. He has already given up a portion of the bone on the other side once. We will take out another piece, because bone reproduces itself all the time. I would suggest taking out another piece at the lower angle, but before we take that out we will expose this tibia and see how the end of the bone is. This is our first opportunity of getting a look at a bone that has been transplanted, because all the other cases have healed, and all have gone to a good and perfect result and there was no excuse to take out the bone. So Dr. Golden will expose the tibia and put in a new transplant, inserting the upper end into the first transplant and the lower end into the tibia.

In making that flap Dr. Golden remembered that primary union of the soft parts is absolutely essential. If you get infection, even of the least virulence, you lose your entire sequestrum. He will not lose it in this case, however, because it is already filled in with new bone. Now, then, the doctor is managing to get his flap out here, keeping on the inner margin of the tibialis anticus, exposing the lower end of the fragment, pulling it up into position, and then exposing the other.

I want to call your attention to the points I made as to diagnosis, because this helps every man. The general practitioner can see it as well as the microscopist, and draw his conclusions from the skiagram. The lobulated condition of that bone is very evident. It is different from the destruction produced by sarcoma of the spindle-cell type.

These bone transplantations have to be done on definite principles, and the definite principles concerning bone transplantation consist of—(*a*) The fragment must be taken out with

Fig. 116.—Skiagram before second operation, showing disappearance of lower end of transplant, due to infection setting in three weeks after first operation.

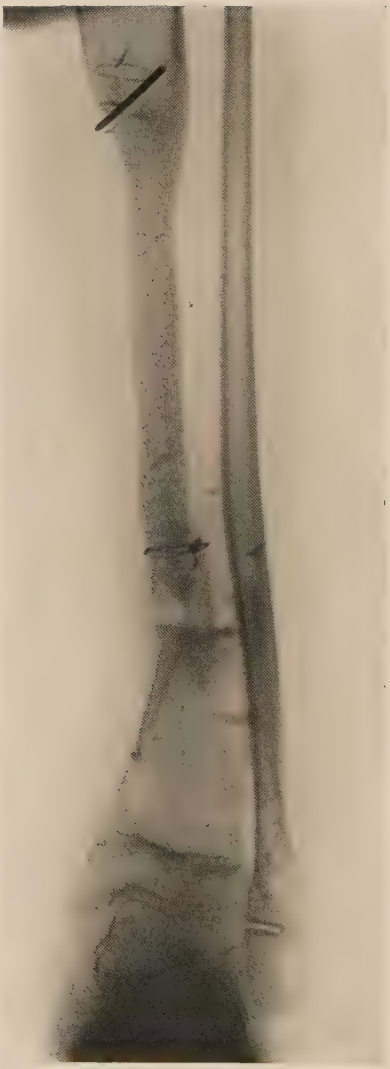

Fig. 117.—Skiagram showing second transplant in place.

or without the periosteum; (*b*) preferably from the same patient; (*c*) if it cannot be obtained from the same patient, it should be obtained, as near as possible, from a patient of the same age; (*d*) when transplanted, it must contact at one or both ends with living healthy bone. It reproduces best when it contacts at both ends. It is better to insert it into the medulla at both ends, but that is not at all necessary. The rôle which the transplant plays is merely that of a scaffold. It always dies. The Haversian canals in the dead bone act as tubes into which the Haversian vessels from the living bone above and below pass. These Haversian vessels from above and below carry with them on their walls osteoblasts and osteoclasts. The osteoclasts dissolve the bone that was transplanted, and the osteoblasts make new bone, so that one brick is taken out and another brick is put in, and long before—months before—all of the transplant is removed you have new tubules in all the old tubules, and firm bony union of the living and dead. There is the interesting thing about it. That continues until all the old bone is removed. New lamellæ are produced around it, and it is all new bone. Then the new bone increases in size and strength as much as is necessary or demanded of it by the extremity.

In a case of osteomyelitis of the tibia in a little girl about eight years of age, the transplant contacted with two little spiculæ of living bone at both ends. The piece we put in measured 5½ by ⅜ by ⅝ by ½ inch. That operation was done in 1908. I saw this patient within the last two weeks. Her leg has grown; she is not in the least lame, and does not know that anything has been done to her leg at all, showing how perfectly the bone can be reproduced. In that case we had to transplant the fibula into the epiphysis of the tibia to prevent shortening of the leg during the period that suppuration was going on. Remember you can never insert a transplant in the presence of pus. Wait until it has entirely healed. That is why Dr. Golden waited so long in this particular case. He waited until it had entirely healed before he put in the additional piece of bone to overcome this separation. It has formed an arthrosis, developing a nice joint. Here is a beautiful joint produced by nature

when we did not desire it, and we took advantage of that knowledge in our arthroplasties. We make new joints readily where the bones of the joint have become ankylosed.

In this case the periosteum is united to the upper fragment. Dr. Golden is peeling off the periosteum from the upper side and he is going to expose the medulla on the lower fragment and take out the fibrous tissue that has formed between the lower and upper fragment, so as to expose the medulla of the lower fragment. Look at this bone—the transplant! See how perfectly the periosteum has grown right to it. Dr. Golden will transplant the piece of bone that he will take from the opposite tibia onto the surface. That shows how nature fills up the gap long before it shows in the skiagram. The skiagram does not show that it is all filled up with new bone, but the gross appearance shows it very well.

Now Dr. Golden is reaming out the medulla in the lower fragment to get room to drive in the transplant. He presses the lower fragment down and the upper fragment up. He transplanted the first piece of bone without the periosteum and it has all reproduced. It is reproduced by Haversian vessels that have gone in from above. This bone will enlarge so that it will be as large as a normal tibia.

Another case we had, similar to this one, except that the disease involved the upper end of the humerus, shows what a detriment the periosteum is to repair. No bone was produced on the white line seen in the skiagram. That is the periosteum which we did not remove from the transplant. Many months afterward the white line is still to be seen, showing where the periosteum interfered with the reproduction of bone. The periosteum is not necessary at all. In analyzing the reproduction and growth of bone I have used this simile in my article in the "Journal of the American Medical Association," namely, that the osteogenetic force is represented by a tree. The trunk of the tree is the medullary canal, which is the one place that retains osteoblasts in the greatest uniformity throughout the entire period of life. The first bifurcations of that tree are the Haversian canals, and next are the canaliculi and lacunæ, and

finally the leaves are represented by the periosteum. Every one of these spaces contains osteoblasts, and every vessel that goes through the medulla carries with it osteoblasts, and they fill up all the transplant with new osteoblasts—first vessels and then osteoblasts—and produce new periosteum with osteogenetic cells.

Here in this clinic we apply our knowledge of the regeneration of bone to five different pathologic conditions: First, in making up for congenital defects, as a flattening of the nose, absence of development of the mandible, failure of development of the radius and ulna and tibia and fibula—never or rarely of the humerus and femur. I have named these aplasic osteal conditions in the order of their frequency. The most common is the nose. In order to fill these spaces you can transplant a bone from any other position in that patient's body that you desire. In making that transplant, whether it is in the nose, in the mandible, in the femur, in the tibia, or in the humerus, you must put the piece that you take from another position in the patient's body in contact with living bone where you implant it. Is that clear? In other words, if you take a piece of bone, with or without its periosteum, transplant it, and, except in very young children, it always dies and fails to reproduce bone and entirely disappears. I transplanted a thumb eight or ten years ago in a case of double thumb; took off the skin and underlying tissues, and put the thumb in to fill a defect in the nose. It healed perfectly and I got very much elated over my success. But in a year and a half the whole thing had disappeared, because I did not take off the periosteum from the transplant, and because I did not contact the bone which I transplanted with living bone denuded of its periosteum, so that the Haversian vessels could get from the living bone into the transplant. If you have that clear you have the whole key to success in bone work, except the mechanical difficulties and the element of asepsis. In doing that work remember that asepsis is absolutely necessary, otherwise a failure occurs. Remember that mechanical exactness of application of bone is necessary, and when you are doing arthroplastic work do not try to do it after reading my article in the "Journal of the American Medical Association." Read over the article if you

wish, and go to the dead-house and do a half-dozen or a dozen operations on the cadaver. In doing that many you will be doing just a tenth or a twentieth of what I did, and then read over my article again and again, and if possible go and see some one do these operations.

I just had the painful experience, within the last forty-eight hours, of telling a very able man that the joint which he made was just as surely going to be a failure as the sun was going to rise, because he did not recognize some of the essential things in doing the work, and Murphy's method will be blamed for the evil results.

Arthroplasties are not easy to do. They are difficult to do; they are not difficult if a man exercises the average intelligence which a cabinet-maker uses or a shoemaker has to exercise before he patches a shoe. A doctor does not possess any superior power, in a mechanical way, to attempt to do mechanical things without preparation more than people do in other walks of life. You can do it. Every doctor should do it. He should give his careful attention to the preparation. I say this today, I think, for the first time, on account of the painful experience I had with an able man in the last forty-eight hours, showing how essential it is to recognize things in detail to get results. Every one who desires to do this work can perfect himself to do it as well or better than I can do it, because it is not difficult if he is prepared for it in a regular and systematic way. It is difficult if you have done no cadaver work and start in by making new joints in the living. You meet obstacles which you will not overcome or even recognize until six weeks after the operation. Then you have spoiled the knee or other joint, and spoiled it for anybody else doing the work.

I want to call your attention to another matter, and that is how perfectly this bone has been reproduced. I want you to look at the thickened new periosteum; how the periosteum has thickened and filled up after the other bone was removed, and how much more vascular it is than the normal periosteum.

That was point No. 1—the treatment of aplasic conditions. Point No. 2 is the good use to which bone transplantation can

be put in securing the reunion of ununited fractures. It is the best and only regular medium of getting union. The mere freshening of the bone following ununited fractures fails in a large percentage of cases.

We had one such case where a Lane plate was used to maintain fixation of an ununited fracture of the humerus. Nothing could be more beautiful than the apposition of the ends of the fragment, and not the slightest effort at the production of bone is evident—a non-union with a most perfect apposition, and that is the most frequent position of non-union in the human anatomy.

In the treatment of ununited fractures, as illustrated in this case, the first thing is to remove the foreign body and clean out the plastic focus and get complete union without infection; then freshen the ends and drill a hole into each one. I have an instrument made especially for this purpose—a medulla drill or reamer. Drill a hole in the medulla and enlarge it to any degree you desire. Then take out the transplant from the crest of the tibia, with or without the periosteum, and drive the transplant into each fragment. Here the transplant was driven down into the lower fragment an inch. Then take a rectangular forceps and drive the transplant into the upper fragment as far as you wish.

In one of these cases we made three skiagrams. Two showed a fairly good union. One showed that there was no union at all, showing the significance of taking pictures at two, three, or four different angles so as to be sure you have all the lines of fracture in the *x*-ray field. Why bone is not reproduced we do not know; whether it is because the fragments are too perfectly immobilized we do not know. You must always take into consideration the class of patients you are dealing with. At the Alexian Brothers Hospital we got the lower class of people, who were overworked and underfed, and it was common to have non-union. The patients at the Cook County Hospital are usually underworked and overfed, and there we very rarely had non-union; but the man who works hard to support his family is always underfed, overstrained, and overworked.

The next class of cases in which you utilize your knowledge of replacing bone that is dead is in fractures of the head of the femur and head of the humerus. We had one case of fracture of the head of the femur. There was rotation of the fractured neck or head of the femur. The head died. We did not see that case until seven weeks after the accident. The head had been dead all that time. We rotated that head around and nailed it to the neck of the bone. Only a little of the head was absorbed. We saw this boy the other day—a week ago last Sunday—and had him walk up and down the corridor before the new interns, and they broke about even at guessing which was the leg operated on three years before. The last skiagram taken just a few weeks ago shows how the epiphysis continues to grow and the nails push further from the joint. The original operation was done in October, 1908. That shows how perfectly that leg has developed, and how that chap has developed into an active young man without a limp.

In a case of fracture and luxation of the head of the humerus we had to take out the head first and detach it. We freshened the distal end of it and then freshened the proximal end of the distal fragment, replaced the head in the glenoid cavity, and nailed it on the distal fragment. All that transplanted head has acted as a carrier for Haversian vessels and reproduced new bone of exactly the same size and shape as the older bone.

The next condition in which we utilize our knowledge of the transplantation of bone is for the replacement of malignant disease or non-malignant disease, as chondrosarcomata. A young girl, about sixteen years of age, had a chondrosarcoma of the upper end of the tibia, almost involving the joint. We took it out, leaving the articular surface of the tibia intact. Then we put in a piece of tibia taken from the opposite leg, and this girl is back on her feet and is having a reproduction of bone of the regular size.

A man had a traumatic chondrosarcoma of his trochanter. We removed the upper end of the femur entirely and implanted a piece of bone from the opposite tibia, suturing the upper end

into the acetabulum. Reproduction of bone progressed rapidly and he has a useful leg.

We never take out the nails which we put in. If you get suppuration or anything around the nail in the process of repair, even pyocyaneus-infection, then you take that nail out, but the great bulk of our patients go away with the nails in and never come back to have them taken out.

There is no question as to what will happen in this case. If it is aseptic, and he has a contacting of the fragment with living bone, only one thing can happen, and that is filling of that fragment with osteoblasts from below, if not from above, with a reproduction of bone from both sides. This will give a good result in this way. The wire which we use in these cases is made of 12 strands of phosphor-bronze, just as thin as the thinnest silk thread.

The operation will be completed in the usual manner, closing the wound with catgut in its deeper layers and horsehair on the surface. A plaster-of-Paris cast, cut with a Gigli saw after it has hardened, will be put on and kept on for four or five weeks. The boy will not be permitted to put his weight on this leg for several months until we are convinced that the bone has regenerated completely.

CARCINOMA OF THE LIP

HISTORY

MALE, aged forty-two, admitted to Mercy Hospital May 13, 1912. Came to hospital because of a growth on lower lip. Family history negative.

Present History.—In October, 1910, patient noticed a lesion resembling a cold-sore on the lower lip, to the left of the median line. It was eroded and raw, and was not benefited by local treatment. He never traumatized the lip, nor has he smoked a pipe for the past twelve years. About four weeks after its appearance the growth was slightly elevated and hard. It increased in size slowly. Two months after the appearance of the growth the patient had two treatments with the electric cautery, two weeks apart. Following this treatment the mass seemed to disappear for three months, when it again appeared and increased slowly in size.

In July, 1911, he had the lesion cauterized again, this time destroying quite a portion of the lip and leaving a cavity. The growth recurred again. A plaster was applied and this was followed by a slough. He later had two more applications of paste. Since the paste treatment the mass has not come above the surface level, but it seems to be infiltrating the surrounding tissues. For two months past there has been a burning, drawing sensation in the old scar, and there is a slight glandular enlargement on the right side. No loss of weight. General condition good.

COMMENTS AND OPERATION

DR. MURPHY: How did it appear, doctor? Was it first a sore the size of a pea, or was it first a scaly lesion with a scab or crust formation? Did that crust fall off from time to time? Did it enlarge? How did it appear?

THE INTERN: The patient described it as having a scaly appearance.

DR. MURPHY: You gave the patient's description. What a difference there is between the doctor's description and the real facts in the case. To get some idea of what the lesion was primarily one must have an accurate description of it, and an accurate description is lacking.

With the exception of the description of the scar, that is about the clinical course of these cases, but that is just what the clinical course in these cases should not be. In every article that one reads on the subject of cancer, epithelioma, or adenoma this statement appears: "If we could get these cases earlier, we would have better results." We are not getting them earlier; in fact, I think we are getting them later.

In cancer the question that first arises—when one analyzes the statistics, the clinical course, and the final outcome of the case—is, how soon after the first erratic epithelial cell penetrates its basement-membrane, which is the one condition that makes it a cancer—how soon after that do these cells pass into the neighboring lymph-spaces, and how soon are they passed on into the first lymph-filter, the first lymph-node? Up to the present time that question remains unanswered; we have no light on it. There is possibly a definite reason for that. Experimental work with mouse cancer has shown that it does not metastasize so frequently as does cancer in the human being. We are not justified in saying that if we had the cases earlier we could get results until we know something about the transmission of the cancer-cell from the primary penetration of the basement-membrane to the nearest lymph-filter.

In a recent article in one of the British medical journals there appeared an analysis of cases of epithelioma of the lip extending over a period of twenty or twenty-five years, and the author endeavored to establish the final outcome of that class of cases. I must say that when I read that article I received a great shock. It was demonstrated, from the cases that could be traced, that 52 per cent. of the patients who had carcinoma or epithelioma of the lip without any demonstrable metastasis at the time of the

operation died a cancer death! In the case before us today the carcinoma was visible from its very onset,—readily seen by physician and layman,—and yet what has been done to lower a 52 per cent. death-rate from cancer?

What is "early" intervention in this class of cases? Cautery and paste! When these cancers are in some hidden zone where we are unable to demonstrate them by symptoms or physical signs until they have developed to a great size, there is some excuse for failure to operate and we know what we can expect as a mortality in that class of cases. But, compared with the cancers that are so easily recognized and yet give so frightful a mortality, why should there be a difference?

There is a difference,—a well-recognized difference,—and that is the redeeming feature. It is based on anatomic reasons. The hidden ones are later recognized, when the lymph-supply of the zone infected by the carcinoma shows evidence of involvement. A carcinoma is or is not dangerous, from a metastatic standpoint, in proportion to the lymph-supply of the zone primarily involved. Carcinoma occurs more frequently in the rich lymph-supply zone than in the lesser lymph-supply zone. In other words, carcinoma occurs always near the active lymph-zone of a viscus, as in the pylorus, the cervix of the uterus, the cervix of the gall-bladder, and so on. Carcinoma rarely occurs in the fundus of the stomach, the fundus of the uterus, and the fundus of the gall-bladder. The richest lymph-supply in all these viscera is found in the cervical portion of each viscus. Carcinoma of the fundus of the uterus metastasizes very late, and frequently does not metastasize at all. If you analyze the statistics of cancer of the fundus of the stomach, you will find that it gives the best final result. Carcinoma of the lesser lymph-supply zones of the large intestine gives the best final results, while carcinomata that occur near rich lymph-supply zones of the intestines, as well as in the cervices of the organs mentioned, give bad final results.

Now let us come back to the lip. The lip is one of the richly supplied lymph-zones. Metastasis should and does take place earlier here than it does in any other position of the body.

How long after the metastasis takes place is it before the carcinoma shows itself? In analyzing carcinoma of the lip we find that there are two distinct types of return: one occurs in the lip itself, in the lymphatics, far from the point of primary invasion, and the other occurs, first, in the lymphatics of the upper triangle of the neck; second, in the deeper structures of the neck, and, third, one commonly overlooked, in the small lymphatics situated just below the foramen mentum. This zone is overlooked in making the examination practically all the time.

In the case of an epithelioma that we consider to be one of the most innocent types of cancerous tumor, the mortality is 52 per cent. where there is no demonstrable metastasis at the time of the primary operation. Added to that frightful mortality is another 24 per cent. of deaths occurring in those cases in which there is no demonstrable enlargement of the gland at the time of the operation. In other words, 76 per cent. of the patients who have a demonstrable enlargement of a gland at the time of operation die a cancer death. Were it not for the difference in the lymph-supply that statement would be so appalling that we would not be looking for the removal of a carcinoma of the pylorus or a carcinoma of the intestine, but the later transmission and the clinical fact that some patients remain permanently cured—true, the number is small—make us still hope to get some results from surgery even in the deeply situated carcinomata.

In the analysis of the next greatest zone of frequency of carcinoma, that of the mammary gland, we find about an equally unpleasant situation. The improved technic that has been adopted in recent years,—first, that of Halsted; second, the splendid technic of Rodman; and, third, the additional advance brought out by Handley,—would tend to make us believe that we could get better final results in the future than we have been having in the past. That appeared to be true for a long time until Halsted analyzed his statistics at the meeting of the American Surgical Association in Cleveland. That analysis certainly did not justify us in cherishing much hope.

In the case before us the scar is the paste scar. On one side there is a little more invasion than on the other. How much of

this is scar and how much is carcinoma we do not know. We have not removed a portion of it. The growth has returned after removal by cautery and paste and is becoming thicker in the middle. We will excise this entire mass, making a V-shaped incision, one limb of the V extending from the angle of the mouth downward and inward to the base of the lip, the other limb extending from about the center of the lip downward and outward to meet the other limb of the V. None of the blood has been admitted into the mouth. We have packed behind the lip with gauze. The ether vapor passes in through a rubber tube clamped into one nostril, so that no blood can pass down into the lungs or stomach. Having excised the tumor-bearing tissue, we will begin our dissection, searching for metastases in lymph-nodes.

We go, first, into the most dangerous zone for the situation of a secondary node, just over the mental foramen, which I can palpate. That node is recognizable, as a rule, by palpation, and we can remove the node and the underlying periosteum. There is some bleeding from the artery in the foramen. I free the lip-flap from the chin, so as to permit it to slide upward to fill in the gap left after the removal of the tumor, when we are ready for the repair. There has been an infiltration here from the necrosis produced by the paste. We can now inspect this zone of the foramen, and I will remove the connective tissue and periosteum down to the margin of the opening, so that there is no chance of leaving the lymph-node. We will carefully examine that tissue to make sure that it is free from carcinoma.

Now we will make a section of the lip, divide it upward and backward so as to make a new lip, and then slide it forward. I am lengthening the internal incision so as to free the mucosa and thus secure more mucosa for the new lip. That is very important. The new lip must be lined with mucosa throughout its entire extent, and skin and mucosa must be approximated accurately. If we do not have ample mucosa, then we have the scar fixed to the bone and there is a tendency to continued irritation and subsequent immobilization of the lip. In the plan of the operation of reconstruction of the lip my attention is devoted to the mucosa primarily because, from the patient's standpoint, that is very much more important than the skin.

The next zone of danger is the lymph-node that is situated in the anterior pole of the submaxillary salivary gland. This node is situated *in* the pole, not *on* the pole. The mouth is closed so there is no bleeding into it, and we are ready to remove the submaxillary lymph-node. That node is scarcely palpable in this case. We know where it is because it is always in relationship to the salivary gland. It is situated in the anterior pole and just a little to the inner side. I am endeavoring to locate it. I can feel it, and, as I pick it up, I can feel that it is resistant to pressure. We will take off a portion of the under part of the salivary gland and with it we will remove that lymph-node. It is separated from the rest of the structure. I have taken it right out of the capsule, and it stands alone and independent of the gland-structure after the capsule is divided. That is the point in which we see the most frequent recurrence of carcinoma of the lip.

How far should we go in the removal of these glands? How far should we go in the removal of the tissues? When you take off a large piece and you succeed, you think you did wisely and well; when you take off a small piece and succeed, you think you did as well; when you take off a large piece and fail, you conclude you did not do well, regardless of the size of the piece that is removed. I think one can say that of carcinoma in every position in the body. How far one should go in the removal of these glands is illustrated by the work of Wertheim, who has had such brilliant results up to date. But remember, that is only *up to date*. Let us wait until another decade has passed; let us wait until the same period of time has elapsed as has elapsed since the Halsted operation was first done, and see where we will arrive, so far as recurrences are concerned.

In the article by Halsted to which I referred a moment ago, there are reported either 111 or 211—111, if I remember correctly—cases, and in 61 of these there were no demonstrable metastases, either macroscopically or microscopically. Deduct these 61 cases, and what was the percentage of mortality in the remaining cases? In other words, what has been added to our results over those which our forefathers obtained when they merely removed the breast? We must deduct the cases with

demonstrable metastasis, because we all know of the splendid work done at Hopkins, and how carefully they examine their specimens. If there were any metastases, in all probability they would have been found.

An analysis of that class of cases disheartened me very much, and an analysis of the more extensive operations done in various portions of the body later has added to my discomfort, and has made the cancer field rather a hopeless proposition to me. There has not been any material improvement in a quarter of a century. I think we can honestly say we have not improved our cancer results in the last quarter of a century.

We will next direct our attention to the anterior angle of the skin-flap. It falls into position easily because I freed both the right and left flaps. I will put in a deep suture as a tension suture, merely to swing that flap into its proper relationship and hold it there without pressure.

We are now ready to proceed with the construction of the angle of the mouth—a very important step. I am shortening the mucosa and the skin-flap of the upper lip, so as to have the mouth open a little farther to the side and give an ample opening, and that is the all-important thing. The redundant skin and mucosa will pull back to position. The same plan is used on the inner side for the approximation of the mucosa far back, so as to keep an ectropium of the lip from occurring. The upper approximation of the mucosa to the lip is done with silkworm-gut. We are getting an ectropium of the mucosa; that is, it is in excess on account of the method in which the flap was prepared. The angle of the mouth is in this manner reconstructed, so that it is practically the normal angle. The external skin-flaps are then united, and the operation is completed. While it is important to remove enough tissue, it is equally important to secure flaps which will permit of accurate approximation without tension or a minimum degree of tension.

How long after metastasis occurs it is before it shows itself in the enlargement of the gland in epithelioma of the lip we do not know. That is one of the things that originally called our attention to the question of how early it does take place. We had in

this hospital a patient we operated on for epithelioma of the lip. The man came back nine years afterward with an enlarged lymph-node—it was in the time preceding the removal of this node—situated under the anterior portion of the submaxillary salivary gland. The question came up, How long had that infection been present in that gland? It had not returned *in loco;* but nine years after the original operation we removed a squamous-cell epithelial growth from this lymph-node, which means, what? That it had been there nine years, because there is no such thing as a primary squamous-cell carcinoma occurring in a gland. Think of it—nine years! And it remained in that position all that time.

The next matter that attracted our attention was that in the carcinomata of the upper abdomen, particularly of the stomach, —and you will remember that attention was called to this fact very early by the late Dr. Fenger,—there occurs early an enlargement of the lymphatics just above the clavicle. In order to have an epithelial cell travel from a carcinoma of the stomach to the gland above the clavicle it had to pass through five filter glands before it arrived in that position. How long was it in arriving at the first, second, third, fourth, and fifth filters? From the time that it reached the supraclavicular space up to the time of the patient's death there was very little change in the gland; how long had the metastasis been in that gland before it attained a size that made it recognizable? That again makes us ask the question, How early does transmission take place? If we could answer that question, we would have a more rational or definite basis on which to base an opinion as to when we remove cancers, early or late.

Our clinical experience has taught us that the smaller the carcinoma, the less likelihood is there to be a recurrence. That is both true and untrue. Take, for example, a carcinoma occurring as a small nodule in a rather plethoric woman, at the age of thirty-eight to forty-two, and remove it. Inside of three months you will have evidences or manifestations of recurrence in many places in the body, and before the patient's death you will have numerous metastases, but not a return *in loco.* So

disheartening is that particular type of case, occurring in a rather stout woman with carcinoma of her breast at forty-three, that I believe these patients will live longer and live better without the removal of the breast.

Where do they recur? To show you how little we know about it and how few direct points of evidence we have, they will recur in the femur, in the spine, and in the brain. I had in this hospital at one time four cases of bone carcinoma secondary to carcinoma of the breast. Two of these patients came to me primarily because of the bone carcinoma. They came because they had trouble in their bones. One of the patients was a girl who, while playing tennis, strained herself in reaching for the ball. The pain in her thigh became very severe, and finally she came because she thought she had a sciatica. In going over her in the regular systematic way we found that she had two small nodules in her breast, one on each side, not so large as an English walnut. The skiagram showed that she had a softened zone in the upper end of the femur, an inch and a half below the tip of the trochanter. It went on to a pathologic fracture, and she went on to a cancer death without the removal of either of her breasts. There was no occasion for the removal of her breasts when she had a metastatic carcinoma in the femur.

One of the other patients came because of pain in the hip-joint which had been called rheumatism. In looking her over we found that she had an aplasia of the left breast. She insisted that the breast had not changed since she was a young woman. The breast was very small and hard. She had never had pain in that breast, and she did not believe that there was anything wrong with that breast. A skiagram showed that she had a carcinoma in the femur just at the base of the trochanter.

A third case was that of a young woman who came in because of a small nodule in her breast. She was forty years of age, robust, healthy, and vigorous. She had an extensive operation performed and in three months she had pain in her spine, low down; then in her femur, and finally had a metastasis in her brain. She died of metastatic carcinoma some nine or eleven months after we first saw her.

The fourth patient was the mother of one of the most dis-

tinguished medical men of this city; she came to us with carcinoma of moderate size in one breast. Her son had recognized the condition practically early. The woman had complained of pain in her back only two or three days before we saw her. While in the hospital we prescribed for her "lumbago." She went on, her spine bent and bent, and she became paralyzed. Another deposit occurred in her femur, and she finally had a pathologic fracture and died from metastases. You can see with that number of cases in the hospital at one time how appalling it is to one when he thinks of these distinct metastases that occur while the lesion is yet comparatively small at the primary location. But you have as a contrast to that another picture—that of the patient who has a small, firm, scirrhous, hard neoplasm, the tissues being coffer-dammed by connective tissue. That coffer-damming connective tissue has contracted and hardened around every erratic epithelial cell. These patients go on for five, six, seven, eight, or even ten years without inconvenience. You remove the tumor by a minor operation on the breast, and the patient remains permanently cured. That shows the greater resistance offered by one class of patients, not a different disease at all in our opinion, but a different resistance to the disease by the individual, and a lesser, weaker, atrophic, contracting, or diminishing, or whatever you care to call it, lymph-supply that comes with age. We know lymphangitis occurs much less frequently as we advance in years than it does when we are younger, with exactly the same cause operating. Of course, we have that demonstration of lesser resistance occurring with other types of infection in which we rarely ever see carcinoma. For instance, in the arteriosclerotic patients there is a different resistance to infections from that found in the average patient in the average condition. But the same question can be put—How soon after the penetration of the basement-membrane are the cells carried either into the blood-current or to the nearest lymph-node, or through the lymph-sieves into the circulation, and then disseminated throughout the body? Until we can answer that question it seems to me we are not going to make great progress as to the definite reason for recognizing carcinoma "early" or "late."

INTESTINAL OBSTRUCTION (SARCOMA OF KIDNEY?).—CARCINOMA OF THE SPLENIC FLEXURE OF THE COLON

HERE is a little child, about five years of age, which we were called to see the other night for intestinal obstruction. The patient did not have a bowel movement for five days. The child had eaten its dinner and went out to play. She came in from play screaming with pain and had nausea and vomiting. The child is pale. It is a typical and classic picture of an intestinal intussusception. The patient was brought in here on the fifth day of the trouble. No bowel movement for five days, and it had vomited only twice, the doctor says. That settled the diagnosis so far as intestinal obstruction was concerned.

In every case of mechanical obstruction of the intestine there is emesis, increasing in frequency, and when the 'phone message came I said, "Has the child vomited?" The doctor said "No, the child has not vomited for three days." It was certain at once that that was not an intestinal obstruction—it was not an intussusception: it was some other lesion. We examined the child, and the abdomen was very much more distended than you see it now, and did not show the beautiful contrast picture. You never see an intestinal obstruction in which half the abdomen is perfectly free except in rotation of the large intestine, occurring in children and adults, with the condition of megacolon. In the condition of megacolon you often see a very large mass in the abdomen. I had one case in which the megacolon contained 56 pounds of feces.

In looking this child over and examining it that night we could get well under an edge, and an edge is always an important thing in the abdomen—a sharp edge. We asked the father if the child had been perfectly well up to this time. At first he said "yes," but finally he said that on two or three different occasions the child had passed blood in the urine. *That told the story.* It was

an acute obstruction—an acute retention with either a cystic or a sarcomatous kidney. We will wait until the acute symptoms subside before we make an exploration.

It seems more like a case of sarcoma of the kidney. There was blood in the urine on two or three different occasions, and hematuria rarely ever occurs in a congenital cystic or acquired cystic kidney in this age. In the case of sarcoma and hypernephroma blood is usually found in the urine. The child is too young to have a carcinoma. There is another type of neoplasm —the endothelioma—which may occur at this age, and which may resemble a sarcoma in its clinical course, but it is a true endothelioma.

The next question is, if this is a case of sarcoma of the kidney, what will you do? Would you say to remove a tumor of that size from a child five years old? The tumor goes clear down into the pelvis, and its removal would involve considerable hazard. No! It is wonderful how well these children stand the removal of these neoplasms, but there is not recorded a single case of permanent cure of sarcoma of the kidney in a child where the diagnosis is unquestioned. In every case, as I told you a few days ago in speaking of sarcoma of the testicle and ovary, there is a recurrence of the sarcoma in the other kidney. Just as soon as you take out the affected kidney a growth rapidly forms in the healthy kidney; whereas if the affected kidney is not removed, the other one may remain quiescent for years or until you disturb it.

I recall a patient who was sent to me years ago by Dr. Earle. I examined him, made a diagnosis of sarcoma of the kidney, and advised against operation. The patient went along for a year and had one or two hemorrhages, but there was no material change in his condition. He went along another year and another year, and finally, at the end of five years, he returned to me. I felt then that this could not be a malignant neoplasm, and that, therefore, it should be removed. I thought that it was probably of the hypernephroma type and could be removed.

We removed the kidney without any particular shock to the patient, though it was larger than the one in this child. On

examination it was found to be an endothelioma, and in ninety days the patient was dead with a similar growth in the other kidney. That gives you an idea of the course of these cases. This man went along for five years with his tumor, feeling fairly well, but when we removed his affected kidney the growth appeared quickly in the other kidney, grew like a mushroom, and killed the patient in ninety days. The moment you throw additional labor on the other kidney it starts up the process and another endothelioma grows in the other kidney and terminates fatally, with alarming rapidity. The patient was never really well from the day he was operated on until he died.

Therefore in the case of this child the only operation that will be done will be just of an exploratory nature. If it is a cystic kidney, then we must carefully examine the kidney on the other side and ascertain whether it is normal, because congenital cystic kidneys are often bilateral, and if they are, one should be very slow to remove the kidney that first attracts attention.

Now, doctor, please read the history of the patient we will operate on now.

HISTORY

Mr. F., aged forty-two, mail-carrier, admitted to Mercy Hospital May 7, 1912, because of pain preceding and during bowel movements. Family history negative. Had gonorrhea at twenty-seven, with orchitis and epididymitis as a complication, also a urethral stricture, for which sounds were passed repeatedly. Denies syphilis. Had typhoid at twenty-one, with good recovery. Was operated on eighteen years ago for inguinal hernia and undescended testicle on right side. The testicle was removed —on account of atrophy, the patient states. Seven years ago an operation was done on his left kidney for stone. Blood in urine was the only symptom preceding this operation. Skiagram showed a shadow of stone in the kidney, but there was no stone found at the operation (done by a prominent surgeon).

Dr. Murphy: That was an undescended testicle, was it not?

The Intern: Yes, doctor.

Dr. Murphy: And should always be removed, because so many of these testicles become sarcomatous.

DR. MURPHY: An additional skiagram was taken, which showed, what?

THE INTERN: A shadow.

DR. MURPHY: Yes, that is an important point.

Five years ago a second operation was done on this kidney. For the past three months the patient has had colicky pain in the left side of the abdomen, of the spasmodic type, and always followed by a bowel movement. Hemorrhages into the bladder stopped after the second operation.

DR. MURPHY: What effect did the removal of the stone have?

THE INTERN: No effect.

DR. MURPHY: Oh, yes it did. My dear doctor, it had the effect of stopping the hemorrhage from his bladder. That is a pretty good proof that the doctor did take out a stone; it relieved him of the hemorrhage, and he has not had any hemorrhage since. But he had pain in his left hypochondrium. Put that in, doctor, because it fills out the cycle as it should be, and throws light on his present trouble, and why we are making this exploratory laparotomy.

COMMENTS AND OPERATION

The history shows that he was relieved of his renal complication at the time of operation, and that he now has a new train of symptoms. First, he has pain in the left hypochondrium; following that pain he has a bowel movement; to use his own expression, he has "a bowel movement with pain." When you analyze that you find that he first has the pain and then the bowel movement. He says, "When I have pain I can always feel a mass in my left hypochondrium."

When we examined him yesterday we kneaded his abdomen and suddenly there appeared a mass. Whether it is a fecal retention or a flexion or fixation of the bowel, from the fact that his kidney has been operated on twice, or whether it is a lesion in the lumen of the bowel, I do not know. But I do know that the cycle of symptoms is the cycle of intestinal obstruction of the obturator type. It is periodic, recurrent, and increasing in frequency. These are the salient things about that history:

First, all his renal symptoms are wiped out by the last operation; second, the urine examination is negative, showing that he has no renal trouble now; third, he has these pains in a definite place—they are of the spasmodic type, and associated with his bowel movements; fourth, when this pain attains its greatest degree of severity he hears a gurgling. That is the return gurgling that comes after a spasm of the bowel, and it is characteristic of that condition.

Now, we will endeavor to find the cause of his trouble on the left side. I could not feel the tumor except after excitation. What it was that produced it we will try and show you today.

I am going to open the abdomen by making the incision at the outer margin of the left rectus muscle, near the linea semilunaris. I will cut through the skin and superficial and deep fascia until the anterior aponeurosis of the left rectus muscle is exposed. This aponeurosis is split so that we can displace the rectus muscle inward and pass through the posterior layer of the rectus sheath into the abdominal cavity. By this procedure the opening in the aponeurosis and that in the peritoneum will not be on the same line. Next, we incise the peritoneum and expose the intestine. We will examine the intestine carefully, searching for the mass which we palpated through the abdominal wall. Here it is in the transverse colon. It is a carcinoma—a carcinoma of the large intestine. Our analysis of the symptoms was correct. We could not feel this tumor until the spasm of the intestinal muscle brought it into prominence. We will do a resection of the bowel and make a lateral anastomosis. The first step is to expose the zone of the tumor in the mesentery. Now we will commence to ligate the mesocolon and expose the zone of the tumor. I am ligating this mesentery, and with a few ligatures above so as to free the upper half and with a few ligatures below so as to free the lower half, from its attachment. We free it for a number of inches both above and below. We will examine the mesentery carefully for enlarged lymph-nodes. There is just one node involved. It contains a large metastatic carcinoma. We palpate the mesentery a little more to see if there are any more nodes involved. There seems to have been

only that one enlarged gland in the entire zone. Now I am in the dead space, through which I will pass my ligature through the mesentery. I will pass along over that zone and get beyond the line of metastases to the next dead space, where I will put on the next ligature. That will allow me to remove about six inches of the intestine. I have to go as far away as that so as to keep away from the vessels.

In operating on the large mesentery always use the angiotribe or crushing forceps. That makes a crease into which the ligature fits, forces out the fat, and when I cut off the mesentery there remains a ledge on the end over which the ligature will not slip. I have in the bite of the forceps practically only blood-vessels and the peritoneum of the upper and lower margins.

I will ligate this mesentery on the distal side, because otherwise I would have a bleeding from that. This is only a temporary ligature, because all of that zone is to be excised.

This patient has had repeated attacks of obstruction, and that has caused the overdevelopment of the muscle layers of the bowel, which caused that tumor which we recognized yesterday, and which appeared after we irritated the bowel and produced contraction of the muscularis. We are now ready to divide the intestine, and the method by which we divide the large intestine is practically identical with the method by which we divide the appendix.

I lift the intestine up into the field and find the portion which I am going to resect. I will put that intestine into this angiotribe; that forces the mucosa of the bowel out of the bite of the ligature, just as in the case of the appendix. Then I divide the bowel with a knife, cauterize and sterilize it, and then invaginate it, just as I would an appendix. With a fine straight needle threaded with silk I pick up the proximal end of the bowel at the mesenteric side first, because that is always the danger-point so far as bleeding, infection, and leakage are concerned, because there is no peritoneum over that zone. I will turn that end into the lumen of the bowel. There will not be bleeding into the bowel. Why? Because I have cauterized the bowel; it will not suppurate at its end because I have sterilized it. This

proximal end is the most dangerous end. Why? Because this leaves a blunt end on the proximal portion against which the pressure of the intestinal contents will come. For that reason we will insert here, in addition to the ligature, two rows of continuous linen sutures. That closes the proximal end of the bowel safely and securely.

In the distal portion there is an excess of tissue; there is our neoplasm. I am going to excise it with about six inches of intestine, but I will first make a lateral intestinal approximation. The line of union will be near the splenic flexure. The splenic attachment is high up, in the usual way. We must free these bands a little and let the bowel come down.

I am going to make the division of the bowel close to the splenic flexure, but I will make the approximation before I excise any bowel. After I have made the attachment I will amputate. There is the omentum coming over from the side; we will later fold that around the line of apposition. I make this union by the regular continuous suture. The angiotribe is not put on the distal portion of bowel beyond the line of division. The first suture which I will pass through here is a continuous suture including only the fibrous or serous layer of the intestine. First we put in the posterior Lembert sutures, then we put in the Czerny sutures without opening the bowel until we have put in the return series of stitches. These two sutures go through all the coats of the bowel and they are inserted parallel to the long axis of the bowel. We pull them fairly taut and place the stitches close together, so as to lessen the likelihood of bleeding into the bowel.

I commence to make my first return stitch, just where I am going to divide the bowel. I am using the Moynihan needle for that. I have completed the Czerny portion of the stitch, and I am going back and do the Lembert portion. The two portions of bowel are then incised so as to establish continuity of the lumen. We complete the anastomosis by continuing the sutures around the openings into the bowel in the usual manner.

Now we are ready for the amputation of the distal end. The intestine is surrounded by strips of gauze and the field of opera-

tion is walled off, just the same as we did at the other end. The clamp is applied and the intestine is severed so that we can now remove the tumor mass. We place a permanent ligature on the invaginated portion in the groove made by the clamp to avoid hemorrhage. This end will have the least tension put on it because it is distal to the approximation and distal to the bowel that will carry the feces on down past the line of approximation. Having completed the intestinal union and securely closed both ends of the bowel, I will fasten the omentum over the line of union with a few fine catgut sutures, so as to anchor it. That gives additional security against leakage and infection. The late Dr. Senn accentuated the importance of this procedure, and I think he was perfectly justified in doing so.

Having replaced the intestine in the abdominal cavity, the next step is to suture the peritoneum by making an ectropium of the cut edges, turning them out and thus preventing the formation of intra-abdominal adhesions. A continuous catgut suture is used for this purpose.

The incision in the posterior and anterior sheaths of the rectus muscle are closed with catgut. The deep tissues of the abdominal wall are approximated and sutured with a silkworm-gut figure-of-8 stitch; the skin is closed with horsehair. The wound is dusted with bismuth subiodid and covered with a gauze collodion dressing and a large abdominal pad over all.

In all these cases we put in a rectal tube. Why? To prevent the accumulation of gas, so there will be no tension on the intestine.

Let us examine the specimen. It is a carcinoma, not a hypertrophic tuberculosis. There are no caseous deposits. There is a stenosis of the bowel. What was the report on the feces examination, doctor?

THE INTERN: Blood was found every time.

DR. MURPHY: Every feces examination showed blood. Let us connect the pathology with the clinical course of the disease. We were told that the stools were formed in a ribbon-like way. That is a misleading statement, because it leads one to believe that there is some obstruction at the outlet. The intern's

version of the case fits an obstruction lower down. When the feces were passed, blood was on the surface, rather than mixed in with the mass. What was said about the pain relative to the bowel movements? The pain was followed by a bowel movement, but the most important thing is that when he had pain he also had the formation of a tumor; he told us the story before we saw the tumor. He had the formation of a tumor in his abdomen on the left side. He said, "When I get a real cramp I can feel the tumor, and as the tumor reaches its greatest size and the pain the maximum degree of severity, I hear the explosion of gas." That is typical; it comes along harder and harder, then finally ceases, and then comes the explosion—you hear a gurgling.

I remember a case which I saw many years ago. We kept the patient under observation for about four weeks, and every day, when we gave her a full meal, the gas came up to the obstruction, and on placing the stethoscope over the tumor one could hear an explosion; the pain would cease, and she would have no more pain until we gave her another full meal. She had these attacks of obstruction in the small intestine for three years. The operation showed an opening or lumen in the bowel a little larger than the lead in a lead-pencil, and an annular stricture of the small intestine just above that point. It was enormously enlarged.

STUDENTS' CLINIC—FRACTURES

[We have received many requests from our subscribers to publish, in one of the issues of the CLINICS, a report of the clinics which Dr. Murphy holds for the senior class of students at the Northwestern University Medical School. In accordance with these requests we publish below a verbatim report of a students' clinic, feeling assured that this will prove interesting to our readers.—PUBLISHERS' NOTE.]

DR. MURPHY: At the last clinic we had under consideration the subject of fractures. What do you understand by a compound fracture?

FIRST STUDENT: A compound fracture is one where the bone fragment penetrates the skin or an opening is made from the skin or mucous membrane to the point of fracture.

DR. MURPHY: And the fractured bone is visible externally or has been retracted inward?

FIRST STUDENT: Yes, sir.

DR. MURPHY: Into what classes do we divide fractures?

FIRST STUDENT: Into simple and compound. A compound fracture communicates externally, directly or indirectly, and a simple fracture does not.

DR. MURPHY: Does that make any special difference?

FIRST STUDENT: It makes a difference in the treatment.

DR. MURPHY: Why?

FIRST STUDENT: Because there is liable to be more infection in a compound fracture than in a simple fracture.

DR. MURPHY: And that infection in a compound fracture is known as what type of infection?

FIRST STUDENT: Direct.

DR. MURPHY: And the infection in a simple fracture is indirect or what?

SECOND STUDENT: Hematogenous.

DR. MURPHY: What do you understand by indirect infection in a fracture?

SECOND STUDENT: Infection through the blood-stream.

DR. MURPHY: How does it get to the fracture?

SECOND STUDENT: There is an extravasation of blood into the gap between the fragments of the bone; bacteria that have gained entrance to the blood-stream are lodged at the site of fracture.

DR. MURPHY: What is that?

SECOND STUDENT: Bacteria gain entrance to the blood-stream and lodge at the point of least resistance or point of internal laceration.

DR. MURPHY: With the extravasation of blood there occurs escape of bacteria from the blood-stream into what?

SECOND STUDENT: Into the clot at the point of separation of the bone.

DR. MURPHY: How can that effect be produced artificially?

SECOND STUDENT: By trauma.

DR. MURPHY: What do you mean by trauma?

SECOND STUDENT: An injury, such as a blow or an operation.

DR. MURPHY: You have not figured that out very well. What have you to say, sir? [Referring to the third student.]

THIRD STUDENT: When one breaks a dog's leg and a nidus of infection has been created somewhere else in his body, there may be bacteria free in the blood and they will lodge at the site of fracture.

DR. MURPHY: Particularly if you do what?

THIRD STUDENT: If you inject bacteria before the injury.

DR. MURPHY: Because they are circulating in the blood?

THIRD STUDENT: Yes, sir.

DR. MURPHY: What filter have they passed through? How do you get infection in a rabbit's leg?

THIRD STUDENT: The bacteria have already passed from the lymphatics into the blood-stream. They have broken through the lymph-glands.

DR. MURPHY: No, not at all; not at all. If you inject bacteria into the blood-vessel of a rabbit's ear, how does the rabbit get an infection in his leg?

THIRD STUDENT: The lymphatics gather up the septic material and carry it to the venous side of the circulation and then

to the arterial side, and from there to the leg. The septic material in the vein of the rabbit's ear passes first into the right side of the heart, then into the first filter, the lung, and if not arrested, into the left side of the heart and on into the general circulation.

DR. MURPHY: And after it passes through the capillaries of the lungs it can be arrested where?

THIRD STUDENT: It can be arrested in the heart, left side.

DR. MURPHY: In what zone?

THIRD STUDENT: In the arterial zone.

DR. MURPHY: In the general circulation zone, is it not?

THIRD STUDENT: Yes, sir.

DR. MURPHY: That is right. If you inject bacteria into the cellular tissue, they are taken up by the lymphatics before they get into the blood-stream, and after they get into the blood they pass through the lung first. Do we have infections of bone occurring in any other way except accompanying fractures?

THIRD STUDENT: Yes.

DR. MURPHY: In what disease?

THIRD STUDENT: In the disease known as acute infective osteomyelitis, tuberculosis, etc.

DR. MURPHY: What is the difference in the process of repair between a simple fracture and a compound fracture, and why?

FOURTH STUDENT: In a compound fracture you have the clot disturbed as it is formed when there is infection present. In a simple fracture, after the extravasation of blood has occurred, fibroblasts begin to form new tissue, new capillaries are being formed, and healing proceeds with the formation of new bone. In a compound fracture there is infection, which prevents healing. by destroying the embryonal tissues as they are formed.

DR. MURPHY: When there is no infection, what is the difference?

FOURTH STUDENT: There is no difference whatever.

DR. MURPHY: What is that?

FOURTH STUDENT: The only difference, then, is the extent of time.

DR. MURPHY: In the infected fracture no clot is formed. There is a difference, then?

FOURTH STUDENT: Yes, sir.

DR. MURPHY: After infection, if a clot forms, there is a dissolution of the clot by the infection, and still, for a time, granulation tissue forms. The clot breaks down entirely, does it not, and escapes as an exudate?

FOURTH STUDENT: Yes, sir.

DR. MURPHY: In time it is walled off?

FOURTH STUDENT: Yes, from the end of the bone.

DR. MURPHY: How?

FOURTH STUDENT: By granulation tissue forming.

DR. MURPHY: Where?

FOURTH STUDENT: At the end of the fragment.

DR. MURPHY: In every injured tissue, in every tissue that is lacerated, there immediately commences what process?

FOURTH STUDENT: The process of repair.

DR. MURPHY: Close to the line of injury or to the line of trauma the tissue becomes necrotized, and the effort at regeneration commences at the line of demarcation between the living and the dead tissue to separate—what?

FOURTH STUDENT: The living from the dead.

DR. MURPHY: Go on, sir.

FOURTH STUDENT: In a simple fracture, after a clot has formed, the capillaries from the Haversian system and the medullary canal and the periosteum bud out to form granulation tissue out into the clot.

DR. MURPHY: And the clots acts as what?

FOURTH STUDENT: As a scaffolding.

DR. MURPHY: Correct.

FOURTH STUDENT: The fibroblasts come in from the periosteum, forming fibrous tissue. Then, about the third week, the osteoblasts come in from the osteogenetic layer of the periosteum.

DR. MURPHY: Of what?

FOURTH STUDENT: From the osteogenetic layer of the periosteum.

DR. MURPHY: The clot is first displaced by what?

FOURTH STUDENT: By granulation tissue.

DR. MURPHY: And subsequently by fibrous tissue—by newly

formed connective tissue. That is what you mean by fibrous tissue. That constitutes what is known as the temporary soft callus. Union of the ends of the bone is effected by the soft callus. And there comes next—what?

FOURTH STUDENT: The process of ossification.

DR. MURPHY: Yes. Where are the osteogenetic factors situated?

FOURTH STUDENT: In the osteogenetic layer of the periosteum and in the medulla.

DR. MURPHY: Let me give you an example, so that you can never forget where the osteogenetic forces are located. In speaking of the osteogenetic forces and where they are situated they may be likened to the trunk, branches, and leaves of trees. The trunk of a tree represents the medulla. It is the most potent osteogenetic force or tissue in the body at *all periods* of life. Next to that come the Haversian canals, which have elongations of the vessels both from the periosteum and from the medulla. Next come the lacunæ and then the canaliculi, and at the ends of the canaliculi you have spread out the leaves of the osteogenetic tree, represented by the deep osteogenetic layer of the periosteum, which in the springtime of life is enormously potent in the production of bone, but late in life has little potency in the reproduction of bone. Therefore the osteogenetic forces are situated, first, in the medulla; second, in the extensions of the medullary cavity, which are the Haversian canals; third, in the lacunæ, which are extensions of the Haversian canals; fourth, in the canaliculi, which are the extensions again of the lacunæ, and fifth, when they come to the surface they spread out under the periosteum,—the deep layer of periosteum,—representing leaves. You cannot forget that. These positions which I have mentioned represent the locations of the blood-vessels from the center to the periphery of the bone. These blood-vessels carry with them always two cell-controlling elements—the osteoblasts and the osteoclasts. The osteoblasts are the builders of bone, while the osteoclasts remove the dead and unnecessary bone.

Now, when a bone is fractured, regeneration occurs from all five of these positions, varying in degrees at different ages: most

potent, at all times, in the medulla; next in frequency and of greatest potency, in youth, in the periosteum; then in the Haversian canals, then in the lacunæ, and then in the canaliculi. That is a simple matter. You surely can never forget it. On the wall of the blood-vessels are osteogenetic cells known as the osteoblasts, and there are also found the osteolytic cells known as the osteoclasts. Therefore, when a soft callus is formed there commences immediately the reproduction of bone proceeding from all five sources I have mentioned, primarily and most readily, as in the growth of the leaf of a tree, from the periphery or the periosteum. Next, in the deposits in the medulla, and then in the intermediate spaces between the medulla and periosteum.

You have, therefore, callus divided into three distinct sets: First, and most early formed, the subperiosteal callus; second, the medullary callus; and third, the intermediate or definitive callus, the one that is to remain. In normal adults, three and a half weeks after the fracture occurs, a bony callus begins to form in the subperiosteal zone; in five weeks it is formed in the medullary area, and in about six and a half to eight weeks it forms in the intermediate zone between the compact bony ends, and that goes on to complete ossification and to the production of a bony callus sufficiently large to give ample support to the weight-bearing force required in that particular bone.

When that is complete, there commences another process—the process of absorption of the excess of callus, and that process of absorption proceeds in exactly the same order as does the process of production of bone. The absorption commences in the periphery, and all the excessive callus is absorbed except where it contacts with other bone tissue and ossification has taken place. Next, the excessive callus in the medulla is absorbed, and there is effected a complete reëstablishment of the medullary canal, and there remains permanently the definitive callus between the compact bony ends.

That is not difficult to understand. It is essential for you to know these elementary principles if you are ever going to understand the process of repair of bone following fractures, if you are ever going to understand the process of regeneration of bone—

Fig. 118.—Photograph showing fracture of ulna and radius, with good union. Little deformity and good functional results. One view.

Fig. 119.—Photograph showing fracture of ulna and radius with good union. Little deformity and good functional results. Another view.

if you are ever going to analyze and explain to yourselves why in certain cases of fracture there occurs union and why in others there is failure of union. It is very essential for you to know these things if you are going to understand why you resort to certain operative procedures to effect union after primary union of the bony structures has failed. All these things must be clearly settled in your minds. In medicine the first question is—why? You cannot forget if you know why. It is your property when you know why. If you do it in a parrot-like way, without knowing why, it is the other man's property and not your knowledge at all.

In the repair of fractures we often meet with beautiful illustrations of the results following a simple fracture, on the one hand, and a compound fracture, on the other. Just look at these two pictures. Here (Figs. 118 and 119) is a simple fracture that has healed with some deformity. There is some deviation in the contour of the bone—just a little, but the patient has a serviceable limb. Healing has taken place by primary bone repair. Here (Figs. 120, 121, and 122) is a picture showing a compound fracture with considerable deviation of the plumb-line. If you analyze this specimen with a plumb-line drawn through the trochanter to the intercondyloid notch in front or behind, you will see that, notwithstanding the enormous deviation shown there, this man had, when he walked, practically a perfectly straight and serviceable limb. Think of it! You see why. Run a line down on the side and see where it strikes. The bone at the union of the fracture falls in front of the line. There is a marked angular deformity. And yet he had a serviceable limb even with the colossal displacement at the bone ends.

Here we have an illustration of the rôle which a separated fragment of bone plays in bridging over the gap or distance between the fragments in a compound fracture. You can see in this case that this fragment was separated from the proximal and distal bone-ends, but it never lost its vitality; it retained its periosteum and lived. Fragments of this periosteum were carried around here in various positions, and that supplied new osteogenetic elements which finally filled in the defect and gave the man sufficient support for his limb, acting as a trestle-work

or resembling the cantilever plan. It shows how potent these osteogenetic elements are, even under unfavorable conditions, namely, those of infection. It must have taken a long period of time to produce that result, as shown by the excessive callus deposited here.

In other fractures, even where the fragments are placed practically in accurate apposition, there is not the slightest effort at the reproduction of a callus. Look at this picture! Note the splendid position obtained by that Lane bone-plate, with perfect apposition, but no effort whatever on the part of—what? On the part of the osteoblasts to reproduce the bone. Contrast these two pictures. See the enormous osteogenetic force exercised in the one, and the absence of the osteogenetic force in the other. In this case it would do no good whatever to put in another plate: you could not get better apposition than is already procured. What you want to do is to stimulate the reproduction of bone; it can be most effectually accomplished by transplanting a fragment of bone from the patient's tibia and inserting it into the medulla, spanning the gap between the separated ends—some 4½ inches in length. Always use an auto-bone graft, as homo- and hetero-transplants are practically always failures.

That bone merely acts as does a blood-clot. For what? For a scaffolding over which the Haversian vessels may pass from above and from below, passing through these canals, and carrying with them the osteoblasts and osteoclasts, which, in time, will remove all that transplanted fragment, but when they do, the fragment will be replaced by another bony framework of exactly the same mold and size. Finally, that new bone will increase in size in its periphery to support any strain that is placed on it. It is really a physiologic hypertrophy. This is one of the natural results of stress placed on any weak point in the body. You see why we put in that bone transplant. You see why you have repair here, and why there is no effort at repair, even when the bone-ends are brought in apposition, without contact of freshened bony surfaces so that the osteogenetic force can be brought into play and do their part of the work.

Fig. 120.—Fracture of left femur (anterior view), showing a fracture with union, but formation of large amount of bony material. Apparently no deformity.

Fig. 121.—Same case (posterior view), with lower fragment pointed and directed slightly away from end of and inward to upper fragment.

Fig. 122.—Photograph showing marked deformity. Same case (lateral view) showing marked deformity, with maintenance of line of gravity so that patient had normal function of leg so far as usefulness is concerned. This deformity was not apparent in the other photographs.

In a compound fracture there follow the outpouring of blood, the formation of a blood-clot, and then the process of repair can go on just the same as in a simple fracture if infection does not take place. Therefore in every compound fracture the first and most important thing is—what? To protect it from sepsis; to protect it from infection. The way in which it is protected from infection is first to neutralize or sterilize the field around the wound with iodin immediately after the injury; second, to place the fragments in the best position you can without opening the wound, if possible.

If you do open the wound to replace the fragments in apposition with the aid of forceps, avoid all additional trauma at the time of the operation, and, above all things, never endeavor to put in a foreign body to maintain immobilization or to secure fixation immediately after the occurrence of a compound fracture. If you do, according to my personal observation, extending over a period of about thirty years, the result, in an enormous percentage of the cases, will be that they will become infected, and eventually you will have to take out the foreign body. It becomes a detriment in place of an advantage.

That is one of the most difficult things for us to learn or to unlearn regarding a compound fracture. A great deal depends on the way you put in the foreign body. That additional handling will enable a sufficient number of the saprophytes and pyogenic bacteria to produce infection locally, and therefore the foreign body is a detriment. On the other hand, if you let the tissues come together with the least trauma, approximate the edges of the wound with suture, if you can, immobilize the leg, and wait until nature heals the soft parts, the chances are that you will get a good result. If, in the case of a fracture of the femur, the fragment should overlap an inch or two, it does not matter much, for you can put on Buck's extension and overcome the shortening. If it is in the lower leg, you can put on a Buck's extension and overcome the shortening. But immobilize the tissues first, and if you keep everything out of the wound,—fingers, instruments, and other foreign bodies,—you will, in all probability, get primary union.

At the end of three weeks, when primary union is complete, if there is still overlapping of the fragments of two or three inches, then, with the field prepared, expose the ends of the bone, put on traction force, after the plan recommended by Dr. Edward Martin, of Philadelphia, elongate the limb to the necessary extent, and maintain approximation, if necessary, with a Lane bone-plate and intramedullary bone splint from his tibia after Murphy, or in any other way you may choose, close the wound, and primary union will take place, the process of bone repair scarcely having been retarded a day. Why? Because the soft callus that has formed during the three weeks you have been waiting is ready to go on to consolidation. The case will terminate favorably without the patient having been exposed to the hazard of an infection which is so common with immediate operative procedures in compound fractures.

It is the most difficult thing—and it has been a difficult thing for a quarter of a century—to keep instruments and fingers out of a compound fracture. In my early experience it was the habit of the average practitioner who saw a case of fracture on the street to run to a drug-store, wash his hands, and stick his fingers in the wound to see how badly the bone was fractured. What followed? Infection!

When I was a surgeon at the Alexian Brothers Hospital the first question asked by the intern was, Has this wound been examined? That is, was it examined before the police brought the patient to the hospital? If not, we could figure that 80 per cent. of the cases would go on to primary union without infection. If the patient had been examined, you could figure that the patient would have 80 per cent. of chances of infection, because the microörganisms were carried into a field whose resistance had been lowered or destroyed by the trauma. The result was that infection followed. Lane's and our experience is that compound fractures should never be primarily plated or wired or nailed or operated.

Case 2.—Before examining this patient I want the intern to read the history of a patient on whom we operated about an hour ago, because the history tells the story. I want you to listen carefully to this history, because it is the story of two lesions

occurring in the same patient, one terminating in a perfectly good, useful joint; the other terminating in a destroyed joint, which I removed this morning.

TUBERCULOSIS OF RIGHT KNEE-JOINT

HISTORY

Mrs. C., thirty-five years of age, married. Entered the hospital because of pain and soreness in the right knee-joint.

Family history negative as to tuberculosis. Has always been perfectly healthy until present trouble.

Menstrual history negative. Was pregnant but once; one child, now three years old; well.

Present Trouble.—In the spring of 1902 she began to experience slight pains in the right knee-joint. She thought that they were neuralgic, and paid no attention to them. Knee was not sore or tender, not swollen, and did not interfere with use of limb in any way. Pains gradually increased in severity, and continued, with intermissions, until five years ago. For days at a time she would be free from pain.

About five years ago pain became quite severe, and the joint became swollen and tender. Not until this time had symptoms increased to such an extent that she thought it necessary to consult a doctor. The knee gradually became very tender, and she was constantly on guard lest she strike it against chairs or other objects. It was not reddened, and was not too sore to prevent it being used in walking about carefully, but after using it a great deal it would be stiffer than usual the next morning. For the past year it has been much worse, and she has been able to get about only by the use of crutches. She can feel a crunching, slipping sensation in the joint on motion. Knee is not ankylosed and never has been. It can be bent, only very slowly and carefully, however, without causing great pain. Joint never has been opened for injection or drainage. Knee never has been injured or had any trauma. Muscles attached to knee are not atrophied. No sinus or discharge of pus has ever been present.

Three years after the onset of the trouble in the knee she had

a sudden, severe pain in the right ankle, and the joint rapidly became swollen, tender, and reddened. She was sick in bed with a high fever (?) for ten days, but had no chills. She does not remember anything in regard to any exposure or lowering of resistance previous to this, but knows that the attack occurred during cold weather. The ankle-joint got well rapidly, with no ankylosis or even limitation of motion. This attack did not influence the symptoms or course of the disease in her knee in any way. Urine normal. Heart negative.

There are two lesions, you see—one an acute lesion, coming on with a high temperature, swelling of the joint, immediate incapacity, progressing to the stage of inflammation for ten or twelve days, then subsiding and entirely disappearing; the other lesion causing swelling, a little at a time, growing a little worse and a little worse, continuing for a period of nearly five years before the patient was materially inconvenienced by the disease in that knee. It was always painful the day after exercise. Finally, there was an acute exacerbation; the process remained stationary for a while, and then there was another acute exacerbation, again remaining stationary. But always what? Always increasing in severity until finally she comes here with cringing in the knee, great pain, walking on crutches, slight motion in the joint.

The diagnosis in the first case was—what? Rheumatism! What is "rheumatism"? What do you mean by "rheumatism"?

STUDENT: An acute inflammation due to some unknown microörganism.

DR. MURPHY: Tell us about it. "Rheumatism" does not mean very much. It is a nice way of expressing what you do not know.

STUDENT: It begins with a slight fever, swelling of the joints, usually one at a time.

DR. MURPHY: Sometimes.

STUDENT: It usually starts in the knee, then goes into the hip, and down to the ankle.

DR. MURPHY: Sometimes.

STUDENT: It may be confined to one joint.

DR. MURPHY: That is correct—sometimes.

STUDENT: There are usually swelling of the joint, redness of the joint, and tenderness. The patient is usually incapacitated.

DR. MURPHY: What is the matter with the patient?

STUDENT: She is sick.

DR. MURPHY: That does not tell the story, sir. What is the matter with the patient? What is really the matter with this patient?

STUDENT: She has an infection.

DR. MURPHY: What do you mean by that?

STUDENT: That some microörganism in the body is causing infection.

DR. MURPHY: But what do you mean by that?

STUDENT: There is inflammation of the tissues.

DR. MURPHY: So is appendicitis an inflammation of the tissues. What is the matter with the ankle? You said the knee was involved in rheumatism. Let us see where we arrive following the average course, and why we conclude erroneously. Tell us about it.

STUDENT: According to the history, there was an arthritis; then later involvement of the bones of the joint.

DR. MURPHY: What do you mean by that? It is not clear. The other gentleman admitted it was an arthritis. It was in the joint. If he did not use that expression, he intended to do so. But you have not added anything material to the case.

STUDENT: There is a history of slow onset, pain, gradually increasing in severity, and later followed by swelling of the knee-joint, with redness.

DR. MURPHY: There was no redness of the joint at any time.

STUDENT: The course leads to tuberculous involvement.

DR. MURPHY: Do you mean tuberculous "rheumatism"? Do you amend the other student's diagnosis? We do not think it was "rheumatism."

STUDENT: The history points to tubercle bacilli lodging in the epiphyseal line, probably in the joint end of the femur.

DR. MURPHY: Have some kind of precise picture of the pathology in that joint. Have some mental picture of it. If

it is rheumatism, where is the lesion? If it is tuberculous, where is the lesion? If it is neither, where is the lesion, and why?

STUDENT: Tuberculous disease locates itself on the joint surfaces of the epiphyseal line.

DR. MURPHY: No, sir, it never does. There is no such place.

STUDENT: The distal portion of the epiphyseal line.

DR. MURPHY: What do you mean by distal portion of the epiphyseal line? I suppose you mean the line of demarcation where the osteogenetic elements are at work in elongating the bone in infancy and childhood up to adolescence. It is the line of division between the circulation of the epiphysis and the circulation of the diaphysis or shaft in the early part of life. It is a trocha, in other words, and of cartilaginous consistence. It is covered above and below with osteoblasts in great number. Now, then, he says this begins on the epiphysis,—the articular surface of the epiphyseal line,—and the epiphyseal line is never on the articular surface. Go on, doctor.

STUDENT: In childhood the tuberculous focus lodges on the shaft outside of the epiphysis.

DR. MURPHY: Yes, on the shaft side? In infancy the epiphysis is cartilaginous. Metastatic infection of any kind never lodges in a cartilage. That is a law.

STUDENT: The tubercle bacillus lodges where there is an abundant blood-supply.

DR. MURPHY: Yes, it lodges where there is a rich blood-supply, and in infancy and early childhood there are many capillary loops on the shaft side of the epiphysis.

STUDENT: The tubercle bacillus lodges and multiplies and gives off toxins which lead to the formation of a tubercle.

DR. MURPHY: The tissue cells multiply and form a tubercle of increasing size. Finally, there occurs an ischemia of the central portion; this dies, becomes necrotic, and a cheesy nucleus is formed. All right, sir, go on.

STUDENT: The tubercle becomes caseous, or fibrous, or calcified. It may break down. Other tubercles may develop. All these may coalesce and form an abscess, which travels in

the tissues in the direction of least resistance. In the mean time there are irritation and swelling, due to the irritation of the synovial membrane and infiltration of the connective tissue.

DR. MURPHY: The swelling is due to what?

STUDENT: Effusion.

DR. MURPHY: As the process nears the surface of the joint cavity it causes effusion into the joint long before the tubercle bacilli arrive in the joint. It produces irritation and congestion, and the joint is often congested weeks and months before tubercle bacilli enter or before the tuberculous focus ruptures into the joint. The patient is incapacitated, usually manifesting symptoms of a severity in proportion to the degree of disturbance caused on the granulating, inflamed focus and by the pressure on the joint surfaces when the patient walks or exercises. Go on, sir.

STUDENT: The abscess travels around the joint.

DR. MURPHY: It does not travel around the joint. If the focus continues to enlarge, it ruptures inside the capsule and into the joint cavity. When doing arthrectomies we find that in the enormous percentage of cases of knee disease the tuberculosis begins as a primary bone lesion, and not as a synovial lesion. It is a tuberculous osteitis, and the joint surfaces become involved later after the rupture of the bone focus into the joint. That is our guide and aid in the treatment.

What good will it do, if you have a tuberculous focus here in the femur or there in the tibia, to treat that kind of a joint by injecting any kind of fluid into the joint? None whatever, as the lesion is then in the bone. If the lesion in the knee is a primary synovial tuberculosis, as it sometimes is in adults and many times is in children, and as it is more commonly in the hip and in the elbow, then you can benefit the patient by the injection into the joint of a fluid that increases the number of polynuclear leukocytes, and that stimulates the production of connective tissue around the infected focus. But in the knee you get practically no benefit at all from such injections because tuberculosis of the knee in adults is so commonly a primary bone lesion. You should know why you are doing certain things

and why you are not doing others. What was the matter with the girl's knee? Why did you conclude it was tuberculosis?

STUDENT: The rheumatism might be a general septic involvement.

DR. MURPHY: What do you mean? What are you talking about? I am not talking about rheumatism; I am talking about the lesion in the knee.

STUDENT: It first started in the bone.

DR. MURPHY: What symptoms did she have in the beginning? Pain on excessive exercise; a little stiffening, more so when the limb had been exercised severely; no swelling or effusion into the joint in the early stage at all, and at the end of five years she developed a partial incapacity. As soon as she began to use the limb and got it accustomed to walking she had no pain at all. But when simply standing, with her leg in certain positions which produced continuous pressure on the bones of the knee-joint, she had pain. That is not rheumatism. What was it?

STUDENT: Tuberculosis.

Dr. Murphy: Where was the tuberculosis? Either in the femur or the tibia. In this particular case the disease began in the internal tuberosity of the tibia. We found there a caseous mass. It finally ruptured into the joint or extended to the synovial membrane, and then got under the margin of the synovial membrane, burrowed in under, and removed all the joint cartilage. When she entered the hospital she had no cartilage at all in the joint. The two raw bony surfaces were being rubbed together, and it is this which gave her such great pain. There was little, if any, effort at fibrosis; little, if any, effort at resistance on the part of her tissues against the destructive process. She was in a lamentable condition. Often you will find in a joint in which tuberculosis has been present for a period of time an enormous quantity of connective tissue, no definite erosion at all, but a mass of fibrous tissue, with great fixation and limitation of motion in the joint. There is no limitation of motion in this case, because there is no tendency to fibrosis, which is the reparative effort made by the tissues against tuberculous invasion. What was the second lesion—the lesion in the ankle? This case

would not give a good result with injections—tuberculin, immobilization, or any other expectant treatment—as the tissues are making no spontaneous effort to overcome the tuberculosis by fibrous tissue production.

STUDENT: It might be the same thing.

DR. MURPHY: Tell me why it might have been so, and why it might not.

STUDENT: The same process is going on there.

DR. MURPHY: Tell me about it. Was it the same process as in the knee-joint?

STUDENT: I think so.

DR. MURPHY: How erratic and inconsistent that kind of a statement is. One was a lesion that took five years to incapacitate the knee; the other was a lesion that took five hours to incapacitate the ankle, and still you say it was the same infective process.

STUDENT: It is only three years ago since the ankle became involved.

DR. MURPHY: Justify your position. The patient was immediately incapacitated. Defend your position. I think you are in error. I know you are in error.

STUDENT: I did not get the point about five years and three years ago.

DR. MURPHY: The patient was immediately incapacitated, as I have said. The patient had a temperature of 103° F., swelling of the ankle-joint, was intensely sick for ten days and then the process subsided entirely and the ankle-joint was just as good as it was before. That is the history, as I remember it.

STUDENT: I would call it rheumatism.

DR. MURPHY (to next student): What is the matter with the patient's ankle-joint.

STUDENT: The patient has some infection.

DR. MURPHY: That does not mean anything to me, sir. What is the matter with the patient? That is what I want to know. I want you to look again, with your intellectual eye, into that ankle-joint and see what has occurred in it. What was the matter with that ankle-joint that gave this train of symptoms, that

passed through this pathologic course, and finally resulting in the perfect joint condition which we have just been considering?

STUDENT: It must be an arthritis or infection.

DR. MURPHY: What do you mean?

STUDENT: There was inflammation of the synovial membrane?

DR. MURPHY: Yes, but what do you mean?

STUDENT: The lining of the joint is inflamed. There is an exudate thrown out on the surface.

DR. MURPHY: So it was in the other.

STUDENT: Later the ligaments undergo dissolution, and possibly the bone-ends.

DR. MURPHY: Based on what?

STUDENT: Based on the assumption that it is an acute infection.

DR. MURPHY: I should say that your statement is based on an absence of an absolute knowledge of the real condition as far as you are concerned.

DR. MURPHY (to another student): What is the matter with the patient?

STUDENT: As near as I can make out there was some infection in the joint itself.

DR. MURPHY: You called it rheumatism. What do you mean by that?

STUDENT: It is a cloak for one's ignorance.

DR. MURPHY: That puts you right. Now you have a basis on which to stand. Your answer is not so obscure as the other gentleman's was. It is just like we define malaria. When a patient has malaise and we say he has malaria, the term covers a multitude of other physical sins, just the same as rheumatism does. But there are definite lesions, and these must be segregated and analyzed into a recognition of the etiologic factors, into a recognition of the tissue that is involved in the disease, into a recognition of the changes which take place in the tissue, and into a recognition of the secondary lesions that result from the infection, and into a recognition of the process of repair and its extent.

It is three years since that infection occurred. The doctor stated in reading the history that he could not locate the primary invasion. Whether you call it "arthritis" or whether you call it "rheumatism," it is a *metastatic infection* from some primary infection which occurred in some other position in the body. That is proposition number one. There is no such disease as acute idiopathic rheumatism; the inflammation in the joint is not caused by a change of the salts or other constituents of the blood. It is an infection—a secondary infection—in all the acute arthritic lesions.

When the infection is of the severe type, it is initiated with a chill, and then it is more commonly located in one, two, or three joints—rarely four. When it is more virulent it may affect a large number of joints primarily, but finally it concentrates its destructive action on an individual joint. If you aspirate the joint immediately after the manifestation of an effusion into the joint, in a large percentage of cases you will find that that fluid gives a negative result culturally, and that is why for years we concluded that this inflammation in the joint was *not of microbic* origin. That deduction is not at all justified by the *fact* that the *effusion does not contain microörganisms.*

We have only to take an analogous condition of infection in the lung which causes pleurisy. Most frequently you do not find the microörganism that produces the infection in the lung in the fluid in the pleural cavity. Why? Because it is not a surface infection; it is a subsurface infection. In a large percentage of the cases of joint infection you do not find the microorganisms in the fluid. Why? Because it is never a *primary surface* infection. Infections do not take place anywhere in the body on the surface of the synovial or serous membranes. They take place in the subendothelial layer or subsurface, in the tissues beneath. The effusion takes place as the result of the *proximity* of that infection to the endothelial surface. Infection is not on the surface of the synovial membrane, but in the tissues of the synovial membrane. It never occurs primarily in the ligaments; it never occurs primarily in the fibrous capsule. After days, and sometimes after weeks, when the multiplication of the micro-

organisms has taken place beneath the surface, the microorganisms begin to appear in the fluid of the joint.

The best example we can use as illustrating the delayed appearance of the microörganisms in a joint is the Neisserian infection. In the Neisserian infection the gonococcus is absent from the fluid in the joint in the enormous percentage of cases of early aspirations. Later in the disease they begin to be present, increasing from the primary findings of 7 per cent. to later findings of 80 per cent. of the cases, showing that the organism comes to the surface after the lapse of time. It is a synovial membrane infection, not a bone infection. Gonococcous infection of the subperiosteal zone occurs very rarely, but in the joint it is a synovial mural infection, and not surface infection.

The other types of infection which we so commonly see are the metastatic infections from the mouth, particularly the fauces, less frequently the tonsils, the upper respiratory tract, the intestinal tract, the urinary tract, the gall-bladder, and the genital tract, especially of the female. These joint infections are all manifestations of a *metastatic deposit* from the primary focus, and never idiopathic rheumatism.

When you get the case early, you have no difficulty at all in locating the primary focus. When you get the case late, you have great difficulty, as in this case, in locating the primary focus of infection. But you know positively that it was not tuberculosis. Why? Because tuberculosis of the joint never incapacitates a patient inside of three weeks. This lesion incapacitated the patient within a few hours. If it had been a tuberculous process, it would have continued for a long period of time, without causing any serious disturbance, the same as the lesion did in the knee. It was an acute metastatic infection, setting up a synovitis. As the tension in the joint subsided the ankle returned to its normal condition. Occasionally, however, it does not do that. When the infection is more virulent, it destroys the synovial membrane and the cartilage and ankylosis results.

The history of this case gives two beautiful pictures—one, a picture of tuberculosis, slow and insidious in its onset; the other a picture of an acute metastatic arthritis, rapid in onset, with im-

mediate incapacity, high temperature, then all the evidences of infection rapidly subsiding, and the patient having a fully restored joint. When the metastatic infection is severe, with high temperature and associated with chill, it is not infrequently of streptococcic origin. That streptococcic infection, if there is effusion, rapidly subsides, loses its virulence, and leaves the joint undisturbed, and *aspiration* or *tapping* without drainage gives full functional restoration if resorted to *early*.

I want to show you a beautiful illustration of metastatic infection to accentuate in your minds how you must look for the primary trouble.

To Another Patient: How long is it since you had trouble with your knee?

Patient: August, 1908. I had boils on my elbow. They began on the third of August.

Dr. Murphy: What next?

Patient: I had a chill on the thirteenth.

Dr. Murphy: Then what happened?

Patient: My ankle commenced to swell.

Dr. Murphy: What next?

Patient: I had fever.

Dr. Murphy: How high was the fever?

Patient: 103.4° F. For ten days following this I was delirious and my temperature went as high as 106° F.

Dr. Murphy: When you came out of the delirium, what did you find?

Patient: My leg was swollen between the ankle and the knee. An abscess formed, and it was lanced.

Dr. Murphy: Then what happened?

Patient: My leg was lanced again and again.

Dr. Murphy: In place of having a coryza or a bronchitis or a pharyngitis, this patient had boils on her elbow, and ten days after the boils appeared she had a chill, pain in her ankle, extending to her knee. She had so much infection that she was delirious, and she woke up finally at the end of another ten days with holes in her leg and pus pouring out. That is just exactly as it occurs in pyemic conditions. She had her metastatic in-

fection first located in the lower end of the tibia, and then it extended to the upper end of the tibia until it involved the knee and extended around the knee, producing finally a bony ankylosis of the knee and necrosis of a large zone in the tibia. She had been immediately incapacitated and has been permanently incapacitated so far as the knee-joint is concerned.

There is no question about the metastatic origin of this trouble. There is no more question about the metastatic origin of rheumatism or arthritis than there is about that. When you analyze these cases you will be surprised. In all the cases except the streptococcic variety ten, twenty, or thirty days from the origin of the primary infection metastasis takes place. In gonorrhea 90 per cent. of the metastases take place between the eighteenth and twenty-second day, showing what a systematic thing it is, how a certain evolution takes place, how it shifts to the joint. It impresses one forcibly with the fact that the primary infection is in some other position in the body. The reason you should know that is because the destruction of the joint will depend on the severity and the type and virulence of the metastatic process. You must begin the day the joint shows evidence of involvement to provide for prophylaxis against an ankylosis of the joint. That is why I wish to accentuate this. These joint infections are metastatic infections; they may rapidly become destructive, depending on the type and virulence of the infecting microörganism.

In this case there is a large osteomyelitic cavity in the bone, and as soon as that lesion is fully healed, we will do the operation for the restoration of motion in the knee. It is not a favorable case in some particulars, while it is fairly favorable in others. She has a movable patella. The patella is not involved in the disease. The infection came up through the tibia. The tibia became agglutinated to the femur, and the infectious material did not get to be free in the joint at all. It burrowed into the femur and nature coffer-dammed it. Every portion of that tibial and femoral articular surface is involved in the bony ankylosis. This is a case in which we will probably do an arthroplasty. On account of the drains in here on the side, and inability to get a soft

tissue flap from the other side to interpose between the tibia and the femur, to do an arthroplasty we will take a flap from the fascia lata and fat over the trochanteric zone, detach it, and place it between the bones to prevent bony union and give her a movable joint.

POTT'S FRACTURE

DR. MURPHY (addressing student): What is the matter with this young lady?

STUDENT: She fell down Saturday night. (This is Monday morning.)

DR. MURPHY: What happened?

STUDENT: She slipped on the sidewalk.

DR. MURPHY: Yes, go on.

STUDENT: She was walking on the street, fell on the right leg, and could not get up.

DR. MURPHY (to patient): Did your foot slide out or did it slide in?

PATIENT: It slid in.

DR. MURPHY: You sat down on it?

PATIENT: Yes, sir. I could not get up without assistance.

DR. MURPHY (to student): Was the foot turned in when she sat down? She could not sit on her feet unless they were turned in—you never get a Pott's fracture when the foot turns in. The foot must have turned out or everted to cause this result.

STUDENT: She has a Pott's fracture.

DR. MURPHY: But what is the matter with her? What do you mean by Pott's fracture?

STUDENT: Eversion of the foot in which there may be a tearing off of the internal ligament.

DR. MURPHY: Of what does a Pott's fracture consist?

STUDENT: It is always produced by eversion of the foot.

DR. MURPHY: What happened next? What breaks when the foot turns?

STUDENT: The internal lateral ligament is torn.

DR. MURPHY: What gives the strong support against the foot turning out? The fibula, is it not? What breaks?

STUDENT: The fibula.

Dr. Murphy: The fibula always breaks in one direction primarily; the foot turns out, and the interosseous ligament holds the fibula to the tibia. The pressure comes on the tip, and the fibula breaks primarily. If the interosseous ligament is strong enough to hold, the fibula breaks higher up. What happens next? Then comes the break of what? The internal lateral ligament, and the tip of the internal malleolus is fractured to a greater or less degree, and as the force continues and the weight of the leg comes on the astragalus, this becomes wedged between the tibia and the fibula, forces its way upward, and splits the interosseous ligament. If the force continues still further, anterior luxation of the tibia into the notch on the top of the astragalus occurs. That is all there is to a Pott's fracture, and unless you know clearly and precisely what constitutes a Pott's fracture, you are going to pay the price by having evil results.

Let us see what a Pott's fracture consists of. First, fracture of the lower end of the fibula. Second, fracture of the tip of the internal malleolus or of a greater portion of the internal malleolus. Third, a tearing of the interosseous ligament by the wedge-shaped edge of the everted astragalus. Fourth, if the force is continued, there is luxation forward on the astragalus of the lower end of the tibia. These are the elements of a Pott's fracture. If you are going to correct and repair a Pott's fracture you must bear these in mind: First, the anterior luxation of the tibia must be reduced. Look at it! See where the head of the tibia is. It is dislocated forward.

Let us reduce it. This is the last thing that occurs in the process of these fractures. Therefore, reduce the fracture in inverse order. First, overcome the anterior luxation. Of course, the first thing is to diagnose that it is present. You know it is present by the great prominence of the anterior margin of the articular surface of the tibia. You know it is present because you are unable to swing the foot forward to a right angle with the leg. The tendo Achillis fixes it so firmly that you are unable to bring it to a right angle. The first thing, therefore, is to reduce the dislocation of the tibia. How do you reduce it? Not by trying to force the foot forward, because that locks the tibia in that notch on the upper surface of the astragalus. Increase the de-

formity by extending the foot, make a skidway of the upper surface of the astragalus on the posterior surface of the tibia, and then bring the foot forward; make traction and flex it, and it comes up to an acute angle with the leg. You know you have reduced the posterior luxation then.

The next step is what? To reduce the eversion of the foot. With the foot at an acute angle to the leg, evert it as far as possible, tract it downward, and *en masse* push it as far to the inner side as you possibly can. It will not go too far because the fibula will stop it from going in by the pressure on the articular surface of the tibia. Then retain it in that position, flexed on the leg, and adducted as much as possible. Why do you keep it turned in so far? Because you wish to have restoration of the interosseous ligament as close as one possibly can. Unless you restore the interosseous ligament you will have a permanent eversion of the foot, because the patient, when he starts to walk, has first a little eversion, and with the interosseous ligament, loose or torn, the edge formed by the junction of the articular surface of the astragalus and its fibular surface forms a wedge which presses up between the fibula and tibia.

When the foot is not dressed in a sufficiently adducted position, that V-shaped upper surface of the astragalus is driven up between the bones.

The method of treatment in this case will be to relax the tendo Achillis, then draw the foot forward as much as it will come, draw the toes up toward the knee, then displace the foot as far inward as it will go, and let the toes come up. The foot will be dressed in that inverted position. This brings the torn interosseous ligament into absolute contact; it keeps the foot at right angles, and the plaster-of-Paris keeps it quiet until bony union of the fibula occurs. If that does not reduce the deformity, make firm extension, put traction on the foot, throw it over, and put a cast on right away. If you will bear in mind what constitutes a Pott's fracture, you will have no trouble in treating it, remembering that the laceration of the interosseous ligament is the most important of all factors, as far as the final result is concerned. If that is restored accurately and firmly, the patient is going to walk square-footed, and the foot will be plumb with the leg.

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